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Pork, loin, roasted, 100 gms.	21.3	22.3	13	170
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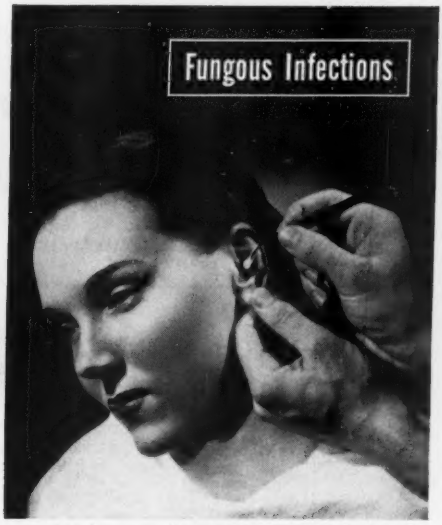

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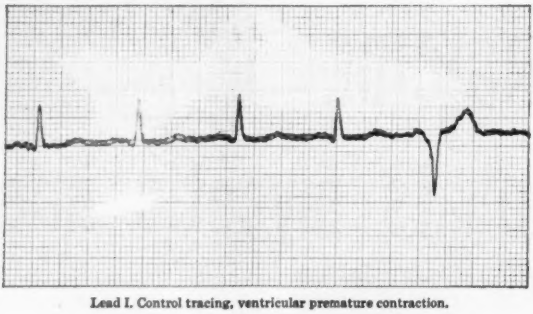
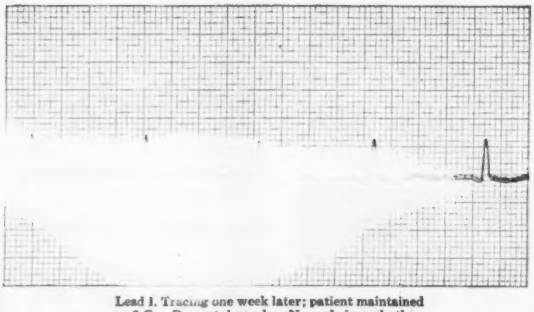


Lead I. Control tracing, ventricular premature contraction.



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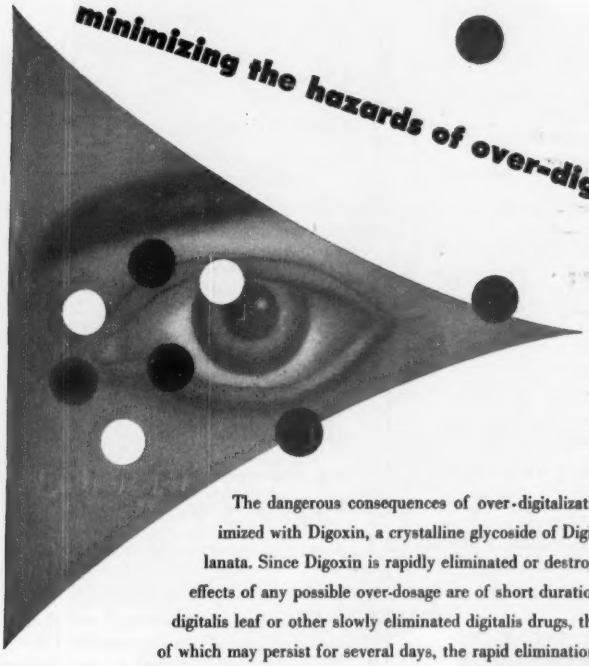

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Volume 244 MAY 17, 1951 Number 20

ACUTE MYOCARDIAL INFARCTION

A Ten-Year Study*

GEORGE E. MACDONALD, M.D.,† AND WILLIAM B. A. BENTLEY, M.D.‡

NEW YORK CITY

THIS report is based on a study of 100 autopsied cases of acute myocardial infarction that occurred during the period of 1940 through 1949. All cases had been admitted to St. Luke's Hospital either because of symptoms referable to myocardial infarction or arteriosclerotic heart disease or for some illness not associated with heart disease per se, in which instance infarction occurred during their period of hospitalization as a major complication.

METHOD OF STUDY

During the ten-year period 1940–1949, a total of 1636 autopsies were performed in this hospital. The protocols of all these patients were reviewed as a preliminary step in selecting cases of myocardial infarction for study. In all cases in which any suggestion of acute infarction was present, in either gross or microscopical post-mortem findings, the clinical records and the microscopical sections were reviewed. As a result, 136 cases were obtained for further evaluation.

Of the 136 cases evaluated, 33 were found to have old infarcts, with inadequate clinical, electrocardiographic and microscopical findings to substantiate the diagnosis of an acute episode. Three cases had clinical histories and electrocardiographic and gross post-mortem findings that were adequate to indicate the presence of acute myocardial infarction. However, the microscopical sections had inadvertently been lost. Since evaluation of the sections was considered one of the most pertinent features of this study, these three cases were not included. One hundred cases remained; these constituted the material for this report.

One of us (W. B.) examined all the microscopical sections of myocardium available in each of the 100 cases (plus the 33 that were found to be "old"). This was done without benefit of the protocol or clinical chart so that he might give an unbiased opinion concerning the duration of the infarct or infarcts in each case. The criteria used by Mallory[1] in estimating the age of an infarct were used. These may be summarized briefly as follows: first twenty-four hours — evidence of necrosis of myocardium characterized by the muscle's taking a deeper stain (hematoxylineosin) with beginning loss of fibrous architecture associated with a beginning peripheral infiltration of neutrophils; leukocytes; two to four days — an increase in the infiltration of these cells; four to five days — beginning peripheral invasion of capillaries, with removal of the necrotic muscle; ten days — peripheral zone of removal of necrotic muscle, with numerous macrophages containing brown pigment, decrease in the neutrophil infiltration and presence of numerous fibroblasts, associated with an infiltration of eosinophils, lymphocytes and plasma cells; twelve days — collagen is present and becomes gradually more prominent by the third week (after the third week accurate dating becomes increasingly difficult because of the slow progress of healing). Infarcts that are completely replaced with collagen and show no evidence of inflammatory reaction, as occurred in 33 cases previously mentioned, are termed "old."

In examining the sections, it was considered essential that the periphery of the infarct with normal muscle should be present, since the changes incident to healing begin at the periphery and progress toward the center. As Mallory pointed out, the microscopical appearance of an infarct and its rate of healing depend to some degree on its size and location and on the status of the circulation in the adjoining myocardium. Accordingly, a large infarct usually heals more slowly than a smaller one and healing is more rapid where there is relatively healthy myocardium adjacent to an infarct than when atrophic or fibrotic muscle is present. These features affect microscopical dating to some extent. For this reason it is important that adjacent non-infarcted muscle be present in the sections examined. For instance, if only the central portion of a large infarct were examined, one would gain the false im-

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†Formerly, resident in medicine, St. Luke's Hospital.

‡Assistant resident in medicine in Department of Pathology, St. Luke's Hospital.

pression that the infarct was more recent than it actually was, because the reparative processes beginning at the periphery of the infarct would be missed.

One of us (G. E. M.) reviewed the clinical records (including the electrocardiograms when taken) and the protocols, assembled the data obtained and arrived at the statistics that will be presented. The following features were included in the study: age of patient, sex, duration of hospitalization, date of onset of symptoms suggestive of infarction, if any were present, clinical diagnosis without aid of electrocardiogram, dates of electrocardiograms, interval between last electrocardiogram and death, electrocardiographic findings (including number of leads employed and diagnosis if made), a re-evaluation of the electrocardiograms, the clinical duration of the infarct, the gross post-mortem findings (including presence or absence of sclerosis of the vessels or recent thrombi or both, location of the infarct or infarcts and rupture of the myocardium), microscopical estimation of the age of the infarct and the correlation between this and the clinical estimate and associated major pathologic conditions.

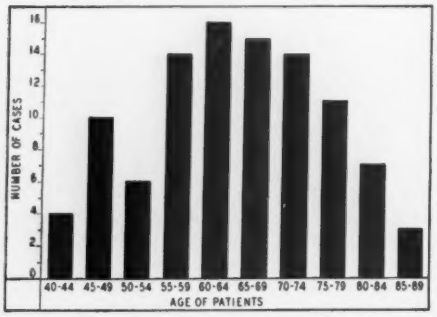

FIGURE 1. *Age Distribution of Myocardial Infarction.*

It must be understood at the outset that we intend to make no generalizations from the statistics that follow. These are the findings in a series of 100 cases of acute myocardial infarction in which post-mortem examinations were performed. When one considers the great number of cases in which no such study could be undertaken because of inadequate clinical and post-mortem data, the limited value of these figures is evident. However, we believe that some conclusions may be drawn from our findings.

PRESENTATION OF CASES

Age of patients. The youngest of the 100 cases studied was 40 years old and the oldest 87. As seen in Figure 1, there was a gradual increase in incidence, with the maximum occurrence of infarcts being found in the 60-to-64 age group. It was interesting to note that 10 per cent occurred in patients 80 to 87 years of age.

Sex. There were 60 men and 40 women.

Period of hospitalization. This varied from one hour to two and a half months. The latter case was included only because of the fact that she presented all the evidence of acute myocardial infarction at the time of admission and, though surviving for two and a half months, suddenly died without post-mortem evidence of any recurrence of infarction. The microscopical sections showed an infarct that was healed and compatible with a two-and-a-half-month duration. This was the only such case included. All others were more recent. Most were present at the time of admission and were so diagnosed clinically; some were present at the time of admission but were not diagnosed until the electrocardiogram suggested their presence, whereas others developed during hospitalization for some allied or unassociated disorder.

Clinical diagnosis. The clinical diagnosis made without the aid of the electrocardiogram was made correctly or considered in the differential diagnosis in 69, or 69 per cent, of the cases. Nineteen per cent of the 100 cases were "silent," leaving 12 per cent in which the diagnosis was missed despite the presentation of complaints and symptoms by the patient, although they may not have been typical in all cases. Of this 12 per cent there were 5 patients who had given a history of chronic intractable angina pectoris, and although the diagnosis of arteriosclerotic heart disease with anginal syndrome was made there was no diagnosis of acute myocardial infarction in these cases. Interestingly enough, the electrocardiograms made at the time of admission in these 5 patients, although abnormal, were not at all pathognomonic of acute infarction. Whether or not acute infarction was actually present at the time of admission will be discussed later.

Electrocardiogram

At least 1 electrocardiogram was made in 85 of the 100 cases. In 15 cases no electrocardiogram was taken, either because the patient died shortly after admission or because the diagnosis of myocardial infarction had not been made clinically. As shown in Table 1, in 52 (or 61 per cent) of the 85 cases an electrocardiographic diagnosis of acute myocardial infarction was made. However, when the date of the last electrocardiogram taken was correlated with the microscopical estimate of the age of the infarct, it was evident that in 10 cases acute infarction had actually occurred after the last electrocardiogram had been taken. This, we believe, is a very important point; it emphasizes the value of determining the duration of the infarct microscopically, as accurately as possible, before attempting any correlation with the electrocardiogram.

Unless this is done, the electrocardiogram will frequently be criticized as not having demonstrated any acute changes when in fact no acute myocardial changes were present at the time the electrocardiogram was taken. For instance, if the electrocardiogram had been taken five days before death and showed no abnormalities and the microscopical connote "healed," which is certainly not always the case). In these 2 cases there were small Q waves associated with inverted T waves without any elevation of the ST segments. In 4 cases, sufficient changes were present in either one or more of the 4 leads taken (standard limb leads plus Lead CF_4) to warrant a recommendation that more leads

TABLE 1. *Electrocardiographic Findings (85 Cases).*

YEAR	NO. OF CASES	NO. OF LEADS	CORRECT DIAGNOSIS	RE-EVALUATION		SUGGESTIVE PATTERNS	ELECTROCARDIOGRAM TAKEN BEFORE INFARCTION	SAME DIAGNOSIS IN RETROSPECT
				ACUTE	RESIDUAL			
1940	10	4	3	3		1	2	1 T-wave changes
1941	15	4	7	1	2	2	2	1 left-bundle-branch block
1942	3	4	3					
1943	4	4	3					1 left-ventricular hypertrophy
1944	9	4	4	1			2	1 left-ventricular hypertrophy 1 residual posterior
1945	8	4	4			1	3	
1946	5	4	5					
1947	11	4	7					2 residual posterior 1 "strain" 1 unsatisfactory
1948	10	*	8					1 left-ventricular "strain" 1 low voltage
1949	10	†	8	1			1	
Totals ...	85		52	6	2	4	10	11

*5	4	Standard limb leads + CF_4.
2	6	Standard limb leads +3 CF leads.
1	7	Standard limb leads +4 CF leads.
1	9	Standard limb leads +6 CF leads.
1	10	Standard + unipolar limb leads + 4 V leads.
†2	7	Standard limb leads +4 CF leads.
2	6	Standard limb leads +3 CF leads.
2	4	Standard limb leads +1 CF lead.
1	9	Standard + unipolar limb +3 V leads.
1	10	Standard + unipolar limb +4 V leads.
2	12	Standard + unipolar +6 V leads.

estimate of the duration of the infarct was two days, one certainly cannot say that the electrocardiogram had been at fault. This seems self-evident, but one occasionally does see reports that attempt to correlate electrocardiographic and post-mortem findings in which the duration of the infarct has not been carefully evaluated. When we then subtract the 10 cases in which the infarction had occurred after the last electrocardiogram, it is seen that a diagnosis was made in 52 of the 75 cases in which an acute infarct was probably present. The percentage of accuracy of the electrocardiographic interpretations is therefore raised to 69 per cent.

In the re-evaluation of the electrocardiograms in each case it was thought that in 6 instances there was sufficient evidence to warrant an electrocardiographic diagnosis of an acute infarct. The diagnoses made instead were "left-ventricular strain" in 4 cases and "myocardial changes" in 2. If, then, we can say that these electrocardiograms were not interpreted correctly, the actual accuracy of the electrocardiograph per se becomes 58 out of 75, or 77 per cent. Of the remaining 17 cases, re-evaluation of the electrocardiograms in 2 cases revealed findings consistent with an interpretation of "residual coronary pattern" (although the term "residual" actually is a poor one, as it frequently seems to be employed, although no diagnosis of infarction could be made from the leads already available. In the remaining 11 cases, the reviewer (G. E. M.) concurred with the original interpretations as listed in Table 1, with the exception of the terminology. We favor "hypertrophy" rather than the term "strain," which connotes to us a functional insufficiency that is not necessarily present.

TABLE 2. *Location of Infarcts.*

	NO. OF CASES	RUPTURE
Anterior	34	3
Anterior and septal	19	2
Anterior and lateral	9	1
Anterior, lateral and septal	3	
Anterior, lateral and posterior	3	
Lateral	4	1
Posterior (left)	17	
Posterior and septal	6	
Posterior and lateral	4	
Posterior (right) and septal	1	1
Totals	100	8

It is impossible to make any definite conclusions from our series in regard to the reliability of the electrocardiogram in proportion to the number of leads employed, since only in recent years have

multiple leads been used in the majority of cases. However, we can make some comment about the reliability of the standard limb leads with one precordial lead (CF_4), which were employed in 72 of the 85 cases. In all 10 cases in which infarction had apparently occurred after the last electrocardiogram had been taken, as described previously, only those 4 leads had been used. Therefore we may disregard them and consider only the cases in which infarction had occurred prior to the last electrocardiogram. In doing this we find that a diagnosis of acute infarction was correctly made in 40 out of 62 cases. From its face value, then, one might say that the electrocardiograph had missed 22 cases. As mentioned previously, these electrocardiograms were reinterpreted, and in 5 instances it was felt that the findings, namely, a prominent Q wave with an elevation of the RT segment in Lead 4, were strongly suggestive of acute myocardial damage. Therefore one might say that the 4-lead electrocardiogram had actually indicated the presence of acute infarction in 45 out of 62 cases (72 per cent). Of the 11 cases in which acute infarction occurred before the last electrocardiogram had been taken and in which an electrocardiographic diagnosis other than infarction had been made — and in which we would make the same diagnosis in retrospect — 1 was diagnosed as left-bundle-branch block, 3 as "residual" posterior patterns, 1 as low voltage, and 1 as technically unsatisfactory. In these 6 instances, it is questionable whether more leads would have added any further information. In the remaining 5 cases in this particular group, augmented leads might have been contributory. In addition to these, there were 4 cases in which enough changes were present to warrant taking more leads. In other words, although the 4 leads were not diagnostic, they were suggestive of a possible infarction.

In summary, it appears that more information was actually obtained from the 3 standard limb leads plus Lead CF_4 than the 72 per cent accuracy figure would indicate. It might be added here that in several of the cases diagnosed with these leads additional leads were subsequently taken. These contributed to estimation of the probable size of the infarct.

From the foregoing it might seem that we are underestimating the value of unipolar limb and precordial leads. Such is not the case. We do believe, however, that when all factors are taken into consideration the 3 standard limb leads plus Lead CF_4 in a fairly high percentage of cases will either indicate the presence of an acute infarct or will show evidence strongly suggestive of it. In any questionable case multiple leads are indicated, whether or not any abnormalities have been demonstrated in these four leads. Of course, multiple leads are of value in localizing an infarct in addition to other information that may be obtained, such as position, hypertrophy and so forth. For this reason we now recommend that augmented unipolar limb and precordial leads be used in all cases clinically diagnosed as probable or possible myocardial infarctions as well as in any other case in which 4 leads have demonstrated some abnormal findings. It is also conceivable that in the middle and older age groups an occasional small silent infarct, such as a directly anterior one, might be missed through taking an insufficient number of leads at routine examination. This seems to be a strong argument in favor of routine taking of multiple leads in all patients over 40 years of age.

Gross Post-mortem Findings

Before discussing the statistics concerning the location of infarcts, recent thrombi and so forth obtained in our cases, we shall review briefly the distribution of the coronary arteries and the changes that may be brought about as a result of collateral circulation developing subsequent to coronary sclerosis.

Although variations or anomalies may occur, the most common findings are as follows:

The left coronary artery, shortly beyond its point of origin, gives off the circumflex branch and continues downward anteriorly as the anterior descending artery, which passes down in the anterior interventricular sulcus to the apex, then continues posteriorly into the posterior interventricular sulcus, extending upward for one fourth to one third the distance to the base of the ventricles. Its branches supply the anterior portion of the left ventricle, the adjacent third of the anterior right ventricle, all of the apical portion of the interventricular septum, the anterior two thirds of the basal portion of the septum, the apex of both ventricles and a variable portion of the posterior aspect of the left ventricle adjacent to the apex. The circumflex branch follows the coronary sulcus, then turns downward to supply as much as half of the basal three fifths of the posterior portion of the left ventricle.

The right coronary artery passes laterally, then downward, in the coronary sulcus, giving off branches that in general run parallel and downward but do not reach the apex. After giving off these branches, it continues to the posterior interventricular sulcus, where it gives off a large branch (posterior descending) that extends along the sulcus for about three fifths of the distance from the sulcus to the apex, penetrating the interventricular system. From the posterior descending artery many branches originate to supply the posterior third of the basal portion of the septum. The right coronary artery continues in the coronary sulcus and crosses the posterior interventricular sulcus to reach the posterior surface of the left ventricle. Here it terminates by dividing into two or more branches, which extend toward the apex but do not reach it.

Anomalies. The circumflex artery may extend to supply all the posterior and basal aspects of the left ventricle, including the posterior portion of the interventricular septum. In some cases it may even extend beyond the posterior interventricular septum to supply the adjoining portion of the right ventricle. The right coronary artery may occasionally extend beyond the obtuse margin of the left ventricle in addition to supplying the apex of the ventricles. Acute occlusion of any of these vessels would ordinarily lead to infarction of the area supplied.

Collateral circulation. Schlesinger has conclusively shown, by means of injection studies, the collateral circulation that develops in most cases of obliterative coronary arteriosclerosis.[2] Although there are normally small anastomoses between the coronary arteries, the diameter of these vessels is only 40 microns or less. Physiologically, therefore, they are unable to counterbalance the ischemia precipitated by the sudden occlusion of an artery. On the other hand, if there has been a slow progressive occlusion of an artery, anastomoses 40 to 200 microns in diameter develop. As a result, complete occlusion of a large vessel may occur without infarction of the area that it normally supplies. Accordingly, as suggested by Schlesinger and Blumgart, the term "myocardial infarction" is more accurate than "coronary thrombosis" as applied to the acute syndrome.[3]

Location of infarcts. The statistics obtained in this particular phase of the study admittedly may be challenged as being open to some question. Over the ten-year period, the post-mortem examinations were performed by many persons. Although a fairly uniform procedure was followed in both performance of autopsy and reporting of findings, there were some differences in the reports, due to the variety of examiners. Some were more thorough than others in writing their reports; some took more sections for microscopical study than did others. In 34 cases, infarction of the anterior wall of the left ventricle (frequently with associated infarction of the posterior apical region) were described, although no mention of infarction of the septum was made. The question might be raised that such a high incidence of anterior involvement without involvement of the septum is unlikely. However, since it is possible for the septum to be spared in anterior infarction it is impossible to discredit any given case as being incomplete. We are aware of this possibility, however. In regard to any statistics based on the microscopical findings, we believe that we are on much firmer ground, since one person (W. B.) had the opportunity to review all the sections taken in each case and did not have to reply on the observations and interpretations of many others.

As shown in Table 2, anterior infarcts were more common than any other. In 34 cases the anterior left ventricle (frequently with associated infarction of the posterior apical tip) was involved alone. In 19 cases the septum was extensively infarcted also. In 9 cases, there was anterior and lateral involvement; in 3, anterior, lateral and anterior-septal involvement; in 3, anterior, lateral and posterior involvement. In 4 cases an acute lateral infarct was noted. In 17 cases there was infarction of the posterior left ventricle; in 6, posterior and septal; in 4, posterior and lateral. There was one acute posterior right-ventricular infarct. This latter case had associated marked obliterative sclerosis of all the vessels with a superimposed recent thrombus in the right coronary artery, with resultant acute infarction and rupture of the ventricle.

In summary, the anterior wall of the left ventricle was described as being involved in 68 per cent of the cases, the lateral wall in 23 per cent, the posterior portion of the left ventricle in 30 per cent, the posterior right ventricle in 1 per cent, the anterior portion of the interventricular septum in 22 per cent and the posterior septum in 6 per cent.

TABLE 3. *Sites of Recent Thrombi.*

	No. of Cases
Anterior descending	21
Right	8
Circumflex	4
Left	1
Right and circumflex	2
Left and circumflex	1
Anterior descending and circumflex	1
Right and anterior descending and circumflex	1
Total	39
	No. of Thrombi
Anterior descending	23
Right	11
Circumflex	9
Left	2
Total	45

Rupture. There were 8 cases of rupture of the ventricle. In 6 instances, rupture occurred through an infarct of the anterior left ventricle; in 1 case, through the lateral wall of the left ventricle. The other site of rupture was through an infarct of the posterior right ventricle, as mentioned above. The latter case was especially scrutinized, since infarction of the right ventricle is unusual in itself and rupture is extremely rare. Sections of the right ventricle showed an infarct seven to ten days old. The left ventricle was described as showing grossly only old healed infarcts. Unfortunately, no sections of the left ventricle were saved.

The duration of the infarcts, as estimated from the clinical and microscopical findings, varied from one to ten days, with an average of 5.6 days.

Recent thrombi. In 39 patients there were 45 coronary arteries occluded by recent thrombi (Table 3). In 21 cases the left anterior descending artery alone was involved; in 8, the right coronary artery; in 4, the circumflex; in 1, the left. In 2 cases there were recent thrombi in both the right coronary

artery and the circumflex branch of the left coronary artery; in 1, thrombi in both the left and circumflex; in 1, thrombi in the left anterior descending and the circumflex. In 1 instance there were recent occlusions of the right, anterior descending and circumflex arteries. On the basis of 45 thrombi, the left anterior descending artery was involved by 51.1 per cent of the thrombi, the right coronary artery by 24.4 per cent, the circumflex by 20 per cent and the left by 4.5 per cent.

Sclerosis. The degree of coronary arteriosclerosis found in these 100 cases has been arbitrarily designated as "marked" if there was involvement of all vessels with marked narrowing of the lumina, "moderate" if there was extensive involvement of the vessels but no uniform narrowing of the lumina and "minimal" if only scattered small plaques were found. Using this classification, 56 per cent showed marked involvement, 34 per cent moderate and 8 per cent minimal; in 2 per cent the vessels appeared normal. In 12 per cent there was apparent obliteration of the left anterior descending artery at some point along its course. There was obliterative sclerosis in 1 per cent of each of the following: right posterior descending, left posterior descending, circumflex and left. Since injection studies were not made in any of these cases, it cannot be stated with certainty whether or not complete obliteration was present.

Microscopical Findings

As mentioned previously, the microscopical findings were considered the most important single feature in this entire study, since evaluation of many of the other data was contingent on the determination of the duration of the infarct. It was possible to make a comparison of the clinical and pathological estimates of the age of the infarcts in 76 cases. In the remaining 24 cases, 19 had been "silent" and 5 patients had had intractable angina for at least several months, so no accurate clinical estimate of the time of infarction could be made.

There was a reasonable similarity in the clinical and microscopical estimate of the age of the infarct in 64 cases (84 per cent). In addition, in 6 other cases the patients had been having symptoms at home for one to three weeks, the symptoms becoming more acute shortly before the time of hospitalization. If we were to assume that until shortly before admission their symptoms had been due to coronary insufficiency and that infarction had then occurred, there would be a close correlation. This is, of course, highly speculative but not unreasonable. None of these 6 patients had had an electrocardiogram taken prior to admission, and in each case acute changes were present at the time of admission. Assuming, then, that this presumption is true, the close correlation is increased to 92 per cent.

In 3 cases (4 per cent) the microscopical estimation of date was earlier than the clinical calculation. In 3 cases (4 per cent), although there was a close correlation between the dating of the initial infarcts, subsequent infarcts occurring during the period of hospitalization had not been detected clinically. Actually, therefore, the close correlation might have been increased to 96 per cent.

As a result, we believe that if close attention is paid to the probable exact time of onset of infarction and the patient is studied closely for evidence of a superimposed acute episode there will be a close correlation between the clinical and microscopical estimates of the age of an infarct. Since this seems to be true, in cases of "silent" infarcts one can calculate from the sections the approximate time when infarction occurred.

Associated major pathologic conditions. The commonest associated abnormalities were marked generalized arteriosclerosis (50), chronic cholecystitis with cholelithiasis (13) and pulmonary infarcts (9). The majority of "silent" infarcts occurred after operations and in patients with cerebral thromboses, cerebral arteriosclerosis, carcinomatosis and so forth, in which cases any symptoms possibly caused by infarction had been masked by the associated disease.

Summary and Conclusions

Data obtained from a study of 100 autopsied cases of acute myocardial infarction that occurred during the period 1940–1949 are presented and discussed.

Without the aid of the electrocardiogram, the clinical diagnosis of acute myocardial infarction was made correctly in 69 per cent of the cases.

Electrocardiograms were taken in 85 cases. An electrocardiographic diagnosis of recent infarction was made in 69 per cent of those cases in which infarction occurred, as dated microscopically, prior to the taking of an electrocardiogram. Re-evaluation of the electrocardiograms in an additional 8 per cent revealed patterns strongly suggestive of acute infarcts; in 5 per cent changes suggestive of myocardial impairment were present; in 7 per cent a "residual" coronary pattern was present. In only 11 per cent no changes suggestive of myocardial infarction were noted.

In 72 of the 85 cases, the 3 standard limb leads plus Lead CF_4 were employed initially. Although there was electrocardiographic evidence of acute infarction in only 72 per cent of the cases in which these 4 leads were used, in an additional 14 per cent abnormal changes compatible with possible infarction were present. In short, in 86 per cent of cases in which only these 4 leads were used the presence of acute infarction was either indicated or suggested.

The distribution of the coronary arteries, changes brought about by obliterative sclerosis and sites of

thrombi, infarction and rupture are described and discussed.

There was a close correlation between the clinical and microscopical estimates of the age of an infarct when Mallory's criteria were used.

Marked generalized arteriosclerosis (50 per cent), chronic cholecystitis with cholelithiasis (13 per cent) and pulmonary infarcts (9 per cent) were the commonest associated major pathologic conditions.

We are indebted to Charles F. Begg, M.D., assistant director of pathology, St. Luke's Hospital, for helpful advice and assistance in the preparation of this paper.

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EPIDEMIC DIARRHEA OF UNKNOWN CAUSE*

Report of Outbreaks in Three Massachusetts State Hospitals

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BOSTON AND WALTHAM, MASSACHUSETTS

ACCUMULATING reports show that epidemic nausea, vomiting and diarrhea were widespread throughout the eastern United States during the period 1946–1947. Epidemics occurred among the populace of Jefferson County, Alabama[1] and in Washington, D. C.[2]; in New York state hospitals[3]; and in maternity services and newborn nurseries in New York City[4] and Washington, D. C.[2] Unpublished reports revealed outbreaks in Annapolis, Maryland, and in boarding schools in New England.

The salient features of those epidemics were described by Fellman in 1921[5] and by Reimann[6] and others.[1-4, 8-10] The disease occurs at all seasons, but epidemics have been reported most frequently in the fall and winter. The etiologic agent has not been cultivated. Dissemination of the agent by common source vehicles such as food or milk has not been reported. Close personal contact appears to be the common mechanism of spread.

Although diarrhea was prevalent in several Massachusetts state hospitals, the purpose of this discussion is to report outbreaks that occurred during that period in three of these hospitals for the insane and mentally defective.

Epidemiology

During the period of December 15, 1946, through February 9, 1947, epidemics of nausea, vomiting, abdominal cramps and diarrhea appeared in three Massachusetts state hospitals for the mentally ill located within the Boston metropolitan area. Vomiting and diarrhea were generally prevalent in that area at that time.

The epidemic in Hospital A began on December 15, that in Hospital B on December 17 and that in Hospital C on January 8. The epidemics, lasting approximately three weeks in each hospital, overlapped in time. Figure 1 compares the shape of the epidemic curves.

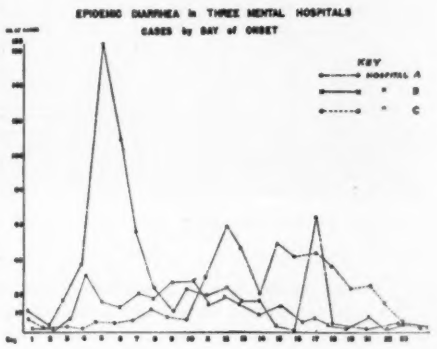

Figure 1.

Within the various buildings and wards of the three hospitals the outbreaks varied from a sharp-point epidemic running its course in five days to a succession of single or multiple cases scattered throughout the three-week period. One building escaped altogether, and certain wards of affected buildings were free of cases. Figures 2, 3 and 4 contain the details of the three epidemics.

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The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

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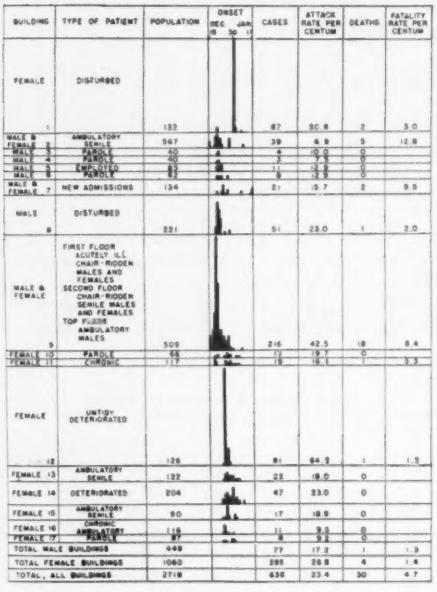
The populations of the three hospitals were 1569, 2113 and 2718. The sexes were about evenly divided. Individual buildings housed patients in numbers varying from 40 to 509. All ages over 6 were represented.

Attack rates for buildings ranged from zero to 79.6 per cent. In general these rates bore no relation to size of population but rather were related,



BUILDING	TYPE OF PATIENT	POPULATION	CASES	ATTACK RATE PER CENTUM	DEATHS	FATALITY RATE PER CENTUM
FEMALE 1	DISTURBED	133	87	80.8	0	3.0
MALE & FEMALE 2	AMBULATORY SENILE	567	39	4.9	5	12.8
MALE 3	PAROLE	40	4	10.0	0	
MALE 4	PAROLE	60	3	5.0	0	
MALE 5	EMPLOYED	82	11	12.8	0	
MALE 6	PAROLE	82	9	12.9	0	
MALE & FEMALE 7	NEW ADMISSIONS	134	21	15.7	2	9.5
MALE 8	DISTURBED	221	51	23.0	1	2.0
MALE & FEMALE 9	FIRST FLOOR ACUTELY ILL CHAIR-RIDDEN MALES AND FEMALES SECOND FLOOR CHAIR-RIDDEN SENILE MALES AND FEMALES TOP FLOOR AMBULATORY MALES	509	216	42.5	18	8.4
FEMALE 10	PAROLE	55	12	19.7	0	
FEMALE 11	CHRONIC	117	19	16.1		5.3
FEMALE 12	UNTIDY DETERIORATED	126	81	64.2	1	1.9
FEMALE 13	AMBULATORY SENILE	122	22	18.0	0	
FEMALE 14	DETERIORATED	204	47	23.0	0	
FEMALE 15	AMBULATORY SENILE CHRONIC	90	17	18.9	0	
FEMALE 16	AMBULATORY	118	11	9.3	0	
FEMALE 17	PAROLE	81	6	7.4	0	
TOTAL MALE BUILDINGS		648	77	17.2	1	1.3
TOTAL FEMALE BUILDINGS		1060	285	26.8	4	1.4
TOTAL, ALL BUILDINGS		2718	638	23.4	30	4.7

FIGURE 2.

with some exceptions, to type of patient. Buildings for the aged, debilitated, untidy and disturbed had the highest attack rates, those for young, active high-grade patients the lowest. Females had higher attack rates than males in two hospitals and a lower rate in one.

Fatality rates for hospitals were 0.57, 2.4 and 4.7 per cent; for buildings, rates varied from zero to 33.4 per cent. In the two hospitals in which fatality rates for sex were available, rates varied between zero and 0.98 per cent for males and between 4.1 and zero per cent for females. The deaths were all among the aged and debilitated.

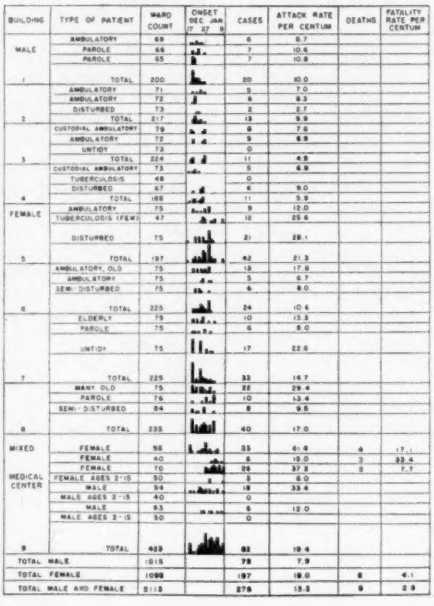
All the hospitals operate central commissaries and kitchens where meals are prepared and from which the food is transported to central dining halls or to diet pantries in the various buildings, where it is served in cafeterias or dining rooms or on individual trays.

In central dining halls, mingling of patients from different buildings is minimized but may occur. Opportunity for mingling is also presented at entertainments and at schools. All the hospitals employ patients for routine work. Thus, although social contact between buildings is limited, patients of one building may work in any other building during working hours, in accordance with their assignments. Such employees care for gardens and grounds, assist in laundries, kitchens, pantries and cafeterias, serve trays, make beds, clean floors and perform other work of similar character.

Each hospital retains one or more buildings for bedridden or seriously ill patients. Minor illnesses are treated in the building of residence.

CLINICAL ASPECTS

The disease in the three hospitals was identical and manifested all degrees of severity. In general



BUILDING	TYPE OF PATIENT	WARD COUNT	CASES	ATTACK RATE PER CENTUM	DEATHS	FATALITY RATE PER CENTUM
MALE 1	AMBULATORY	69	6	8.7		
	PAROLE	66	7	10.6		
	PAROLE	65	7	10.8		
	TOTAL	200	20	10.0		
2	AMBULATORY	71	5	7.0		
	AMBULATORY	72	6	8.3		
	DISTURBED	73	2	2.7		
	TOTAL	217	13	5.9		
3	CUSTODIAL AMBULATORY	79	6	7.6		
	AMBULATORY	72	5	6.9		
	UNTIDY	73	0			
	TOTAL	224	11	4.9		
4	CUSTODIAL AMBULATORY	73	5	6.9		
	TUBERCULOSIS	46	0			
	DISTURBED	67	6	9.0		
	TOTAL	186	11	5.9		
FEMALE 5	AMBULATORY	75	9	12.0		
	TUBERCULOSIS (FEMALE)	47	12	25.6		
	DISTURBED	75	21	28.1		
	TOTAL	197	42	21.3		
6	AMBULATORY, OLD	75	13	17.6		
	AMBULATORY	75	5	6.7		
	SEMI-DISTURBED	75	6	8.0		
	TOTAL	225	24	10.6		
7	ELDERLY	75	10	13.3		
	PAROLE	75	6	8.0		
	UNTIDY	75	17	22.6		
	TOTAL	225	33	14.7		
8	MANY OLD	75	22	29.4		
	PAROLE	76	10	13.4		
	SEMI-DISTURBED	84	8	9.5		
	TOTAL	235	40	17.0		
MIXED MEDICAL CENTER 9	FEMALE	98	33	41.4	4	17.1
	FEMALE	40	6	15.0	2	33.4
	FEMALE	70	26	37.2	2	7.7
	FEMALE AGES 2-15	50	3	6.0		
	MALE	54	18	33.4		
	MALE AGES 2-15	40	0			
	MALE	83	6	12.0		
	MALE AGES 2-15	50	0			
	TOTAL	433	82	19.4		
TOTAL MALE		1018	78	7.9		
TOTAL FEMALE		1098	197	18.0	8	4.1
TOTAL MALE AND FEMALE		2118	275	13.2	9	2.9

FIGURE 3.

it was mild and afebrile; many persons affected were not disabled. The onset was usually abrupt, with nausea, and with vomiting or diarrhea or both. The diarrhea was copious, fluid, brown in color and of foul odor. Abdominal pain varied from mild to painful cramps. The duration in mild cases was twenty-four to forty-eight hours. Convalescence was rapid.

Moderately severe cases had fever ranging as high as 102°F. and marked aggravation of the other symptoms. Not infrequently the stools contained blood after the first few hours of diarrhea. There was a distinct element of toxicity, and in some instances patients became rapidly prostrate. Duration was several days to a week.

The principal feature of the severe cases was the acute shock that occurred soon after the illness was recognized. A few patients had fever, but dehydration and collapse were more prominent. Hematemesis and rectal passage of frank blood were observed. Those patients required intravenously administered fluids and plasma in adequate amounts. The deaths occurred in this group. If death did not occur during the collapse stage, recovery ensued as with the moderately severe cases.

A few patients manifested a subacute form of infection. After the diarrhea ceased, bloody stools appeared that lasted for months in some cases. Proctoscopic examinations revealed marked congestion of the rectal mucosa. Exploratory operation was considered on occasion. However, the bleeding

BUILDING	TYPE OF PATIENT	POPULATION	ONSET JAN. 23 FEB.	CASES	ATTACK RATE PER CENTUM	DEATHS	FATALITY RATE PER CENTUM
MALE							
1	LOW GRADE CONFINED	149		68	45.6	2	2.94
4	AMBULATORY WORKING	62		12	20.6		
5	AMBULATORY WORKING	110		39	35.4		
10	HIGH GRADE SCHOOL	81		34	42.0		
11	HIGH GRADE	103		11	10.7		
13	DISCIPLINARY	29		12	41.4		
14	LOW GRADE INFIRM	74		35	47.3		
15	LOW GRADE INFIRM	174		95	54.6	1	1.05
FEMALE							
2	LOW GRADE CONFINED	179		57	31.8		
3	LOW GRADE, CHRONIC	25		3	12.0		
6	AMBULATORY WORKING	125		31	24.8		
7	HIGH GRADE SCHOOL	93		31	33.4		
8	HIGH GRADE	97		15	15.5		
9	HIGH GRADE	83		5	6.0		
12	HIGH GRADE	40		0	0		
16	LOW GRADE INFIRM	49		39	79.6		
17	LOW GRADE INFIRM	116		36	31.0		
TOTAL MALE		782		306	40.2	3	0.98
TOTAL FEMALE		907		217	26.6		
TOTAL MALE AND FEMALE		1689		523	33.3		0.57

FIGURE 4.

eventually stopped in all instances, and there were no recurrences.

After the epidemic the disease apparently became endemic. It was noted that newly admitted patients became ill whereas none of longer residence were affected.

Stool cultures and paired serums of patients and food handlers revealed no evidence of infection with any of the usual enteric pathogens.

Forty-one patients died. The sexes were about evenly divided. Thirty-two were over 59 years of age, of whom 10 were enfeebled by senility, 4 had cardiac decompensation, 12 had generalized arteriosclerosis and the remainder were affected by various general or central-nervous-system diseases. Seven were between 49 and 60, of whom 3 had general paresis, 1 had paranoia, 1 dementia praecox, 1

FIGURE 5. *Case 3. Small Intestine (Hematoxylin and Eosin Stain x120).*

The section shows necrosis of the epithelial cells, congested capillaries, dilatation and stasis of the vessels of the submucosa, and focal, spotty accumulations of leukocytes.

diabetes mellitus and 1 myotonia dystrophia. There was 1 death at age 34, that of an idiot with epilepsy and severe deterioration. The duration of illness in fatal cases ranged from one to thirteen days, with an average of 4.95 and a median of 5.5 days.

CASE REPORTS

CASE 1. M. E., a 66-year-old depressed, co-operative, deluded woman, was admitted to the hospital building of Hospital B on February 9, 1947, and was isolated for diarrhea. She was treated with Kaomagma, sulfadiazine and penicillin. The diarrhea ceased on the second hospital day and did not recur.

CASE 2. F. P., a 59-year-old man with agitated depression, was housed in the medical building of Hospital A. On December 27, 1946, he developed a severe diarrhea. He was isolated and treated with camphorated tincture of opium and bismuth subcarbonate. Because he was dehydrated, a hypodermoclysis of a 5 per cent solution of glucose was adminis-

tered. The diarrhea continued and by January 4, 1947, he had grown very weak. During the next few days there was slow improvement, and diarrhea ceased on January 24.

Case 3. H. P. S., a 70-year-old man with alcoholic psychosis and general arteriosclerosis, had been living in the medical building of Hospital A, spending all his time in bed or sitting in a chair because of polyneuritis. On December 20, 1946, he became ill with colicky abdominal pain, burning sensation in the throat, vomiting and diarrhea. He vomited dark coffee-grounds material and had 15 to 20 loose, brown, watery, foul-smelling stools a day. He was given large amounts of parenterally administered fluids, Kaomagma and sulfadiazine. Despite treatment the symptoms continued; the diarrhea did not stop, and he vomited everything taken by mouth. He became very pale on the second day of illness, his eyes became sunken and his course was steadily downhill. He was comatose on the fourth day, respirations were rapid and shallow and the stools contained blood. Death occurred at 2:45 p.m. on December 24. The clinical diagnosis of the final illness was acute enteritis and bronchopneumonia.

The important autopsy findings were limited to the lungs and intestines. There was bronchopneumonia at the base of the right lung. There was slight congestion of the mucosa of the duodenum and of about one third of the jejunum. In the lower third of the jejunum the mucosa was dark red and velvety to touch. The ileum was so congested that it looked purplish-red. The cecum had the same color, the appendix seeming pale by comparison. The ascending, transverse and descending colon and sigmoid had a purple-blue-red color. This intense severe congestion ended at the sigmoid, the rectum being merely dark red. The congestion of the mucosa of the ileum and colon was of a definitely hemorrhagic type. The mesentery also contained vessels that were injected but not to the intense degree seen in the bowel. There was no hypertrophy or hyperplasia of the mesenteric lymph nodes.

Microscopically the duodenum showed dilatation and stasis of the vessels of the submucosa; the jejunum showed dilatation of the vessels of the submucosa, greatly congested capillaries of the lamina propria of the villi, infiltration of the submucosa with polymorphonuclear leukocytes and focal spotty accumulations of leukocytes in the muscular coat (Fig. 5). Many of the epithelial cells of the distal third of the villi appeared dark and overstained, and some had necrotic tips.

Pathological Findings

Of the 30 deaths at Hospital A in which enteritis was considered a main or contributory cause of death, 6 autopsies were performed. At the same time 1 autopsy was performed at Hospital B and 2 were performed at a fourth State hospital, where a similar epidemic was in progress, so that the group under discussion consisted of 9 cases. Five were men; 4 were women. The nutritional state was good in 4 and poor in 5 cases. The intestinal contents consisted of blood-stained fluid, and in one case the jejunum contained dark-red hemorrhagic fluid that looked like venous blood. The mucosa was reddish and definitely hemorrhagic in most cases. Mesenteric vessels were congested, although not to the extent seen in the intestinal mucosa, and contained no thrombi. The microscopical findings were more striking in some of the cases than in others, but all showed dilatation and stasis of capillaries and veins of the mucosa, with an early thrombus in one case that was associated with diffuse hemorrhages in the submucosa, and necrosis of the mucosa. Inflammatory cellular infiltrate extended from the mucosa into the submucosa. The infiltrate consisted of polymorphonuclear leukocytes, lymphocytes and monocytes but on the whole was not extensive. The degree of congestion varied and was striking in two cases. Cloudy swelling of the convoluted tubules, with albuminous casts and exudate into glomeruli, was present in some cases. The heart in the older arteriosclerotic patients showed coronary changes, with or without occlusion. The lungs showed bronchopneumonia in 4 cases, pulmonary edema in 2 and congestion in 3. The liver showed chronic passive congestion, central necrosis and some fatty degeneration in the arteriosclerotic patients; in these patients the pancreas showed congestion and overflow of vessels in 1 instance and the spleen showed diffuse hemorrhages in 3 instances. The brain showed typical arteriosclerotic changes in 5 cases; a typical senile atrophy of the brain, with numerous senile plaques and Alzheimer's fibrillary changes, was found in 1.

It was believed that exhaustion and dehydration following enteritis, together with some toxic damage to the kidneys, were contributing factors in these deaths.

Discussion

There seems little reason to doubt that the outbreaks in the three hospitals were due to the same, or similar, agents and that the agents were present in the community at large. From case to case, building to building and hospital to hospital, the clinical features were well defined. Stool cultures and serologic tests revealed no known agent of enteric disease.

In none of the three hospitals was it possible to support the postulate that the responsible agent was spread by food, milk or water from a common source. The water was from chlorinated municipal supplies, milk was pasteurized and food was issued from central kitchens. The buildings were affected on different days; one building escaped altogether. Even in affected buildings, entire wards escaped the disease. However, the rapidity with which the condition spread leaves no doubt of its communicability.

It is most likely that spread was by close personal contact. Opportunity for such transmission was afforded through the medium of patient-employees whose duties took them from their own dormitories into all the hospital buildings and brought them into close association with the other patients. It is of interest that the patient-employees in the building that escaped infection had been confined within it during the outbreak. Assemblies such as entertainments, church, school sessions and meals offered opportunities for contact spread, except among the patients of the building that escaped, who declined such exposure.

The extreme rapidity with which high peaks of incidence developed in certain wards suggested spread by the respiratory route. Reimann and his associates[9] produced gastroenteric symptoms in 17 of 32 human volunteers by spraying into their throats filtered garglings of patients ill with similar

symptoms; they also demonstrated the infectiousness of fecal filtrates. In the present instance both mechanisms could easily have been operative.

The experiments among human volunteers carried on by Gordon and others[10] in New York state hospitals in 1946 and 1947 suggested that an active immunity, probably short-lived, develops.

Sporadic cases, as noted by Reimann,[7] were observed among newly admitted patients after these outbreaks. Whether the agent was disseminated by carriers during the endemic period or was present elsewhere, as in dust, was not apparent.

The disease was not limited to a particular age group; it occurred among patients from 6 to 84. Abramson and Fuerst,[4] in 1948, supplied evidence that completes the description of susceptible age groups. They recorded an outbreak in August, 1946, on the maternity service of a large general hospital in New York City in which the source was a mother in an obstetrical ward; the infection spread rapidly to the mothers in the ward and to babies in the nurseries and then to maternity-service personnel; it subsequently spread to inmates of a child-care institution and to the residents of a tenement house in the community. Similar observations were made in the Boston metropolitan area when a series of outbreaks occurred among newborn infants in several hospitals during the time of the epidemic in the three hospitals under discussion.

Cumming[2] reported introduction of epidemic diarrhea into nurseries by two-day-old infants of mothers convalescent from "contact diarrhea" that was prevalent in the general population of Washington, D. C., in the vicinity of the hospital.

No definite estimate can be made concerning the incubation period from the data of this investigation. The extreme rapidity with which the peak succeeded the first cases in certain of the wards and the equally rapid decline in incidence suggest that it was very short. In their human volunteer experiments Gordon and others[10] found the mean period for 10 artificially induced cases to be three days, with a range of one to five days. Reimann and his co-workers,[9] studying human volunteers, found that the period varied from one to twenty-one days. Abramson and Fuerst[4] estimated the median incubation period at a maternity service and in an infants' home to be two to three days with a range of one to twenty-one days. Smillie and others[1] found it to be one to twelve days in forty cases, with a median of three days. The series of nursery outbreaks observed by Cumming[2] began in two-day-old infants.

The experience of the others supports the fact noted in this epidemic, namely, that treatment is limited to supportive measures. Mild cases require little attention, but moderately severe cases must be watched closely for early signs of shock, collapse or dehydration and must be treated with parenterally administered fluids and plasma in generous amounts. Bleeding in patients surviving the period of collapse has not been severe enough to require transfusions. The outlook for the aged and for invalids is grave in the face of severe symptoms, for their course is often characterized by early collapse leading to death, in spite of the most solicitous care. To date, sulfonamides and the antibiotics have produced no marked effect on the progress of the illness, and the only prophylactic measure available to the aged is isolation from milder cases in the vicinity. It is not clear whether or not infants may suffer from the severer forms of the disease.

Summary

The data describe epidemics of acute enteritis of unknown etiology that spread slowly through the buildings of three large hospitals for the mentally ill located in the Boston metropolitan area.

The pathogenic agent was not identified. The management of the hospitals and the pattern of spread were not such as to support the postulate of a common source or vehicle. On the other hand, the clinical and epidemiologic aspects of the illness seem to indicate that it was a communicable disease. The rapidity with which it attacked closed groups suggests the possibility of spread by the respiratory as well as the intestinal route.

The high mortality noted among elderly and debilitated patients was a striking observation. Prompt institution of therapy in such cases is urgently necessary in order to prevent death.

Pathological examination revealed marked local effects in the mucosa, submucosa and mesentery of the small and large bowels and general effects reflected in cloudy swelling of the renal tubules and, in the case of arteriosclerotic patients, in congestion and hemorrhages in pancreas and spleen, and in congestion and inflammation in the lungs.

Conclusion

The evidence indicates that the three outbreaks were part of a single epidemic in the Boston metropolitan area and that they were examples of the recently recognized epidemic enteritis of unknown (virus) etiology that has been reported from several states in 1946 and 1947.

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REACTION OF THE ADRENAL CORTEX TO PHYSICAL AND EMOTIONAL STRESS IN COLLEGE OARSMEN*

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CHANGES in the level of circulating eosinophils after administration of adrenal cortical hormones, ACTH or epinephrine have been accepted as an index of adrenal cortical activity.[1] Hormones elaborated by the adrenal cortex are known to be links in the physiologic response to stress. The eosinophil response to actual stress situations such as hypoglycemia, surgery, trauma and others[2-4] has been described. The stress situations studied, however, have been as a rule abnormal and artificial.

The Harvard–Yale four-mile boat race and the preceding two weeks of intensive training of crews presented an opportunity to study the response of healthy young men to a standard physical stress. Since the race takes place at the end of a long period of training and rowing, it could also be assumed that the crews were physiologically well prepared. The comparison of the results obtained during and after the actual race with those obtained at time trials during the training period permitted an evaluation of emotional stress in addition to the basic physical exertion. Coxswains and coaches, on the other hand, were submitted to emotional stress only. The almost ideally regular mode of life maintained during the entire period of observation offered a well controlled background for these studies.

During the two weeks preceding the race the competing crews lived in training quarters at some

TABLE 1. *Eosinophil Counts Obtained in the Harvard Varsity Crew.**

DATE	BLOOD SAMPLED	No. 1	No. 2†	No. 3	No. 4	No. 5‡
6/9	Control	68	—	78	68	152
6/10	Control	96	124	80	192	264
6/11	Just before	140	69	34	152	172
First time trial	1 hr. after	48	68	38	86	102
	4 hr. after	14 (−90%)	32 (−55%)	18 (−45%)	14 (−93%)	24 (−86%)
6/12	24 hr. after	163	221	100	131	268
6/14	Control	116	120	44	151	238
6/16	Control			14		
6/17	Just before	118	118	12	129	267
Second time trial	1 hr. after	112	82	7	88	104 (−74%)
	4 hr. after	27 (−77%)	46 (−61%)	6 (−50%)	21 (−83%)	141
6/18	24 hr. after	110	92	41	148	218
6/20	Control	129	96	58	150	163
6/22	Control	124	156	153	173	314
6/23	Just before	122	82	68	101	62
Race	1 hr. after	3 (−98%)	51	0 (−100%)	27	14
	4 hr. after	7	3 (−96%)	—	3 (−97%)	0 (−100%)
6/24	24 hr. after	88	85	—	140	177

*Expressed in eosinophils per cu. mm. of blood.
†Had hay fever.
‡Had hay fever; had undergone splenectomy four years previously.

TABLE 2. *Eosinophil Counts Obtained in the Harvard Combination Crew.**

DATE	BLOOD SAMPLED	No. 1	No. 2	No. 3†	No. 4	No. 5
6/15	Just before	140	123	343	156	98
Time trial	1 hr. after	73	73	274	148	116
	4 hr. after	56 (−60%)	41 (−67%)	175 (−49%)	56 (−64%)	122 (+20%)
6/16	24 hr. after	107	102	314	140	101
6/20	Control	142	54	210	188	62
6/22	Just before	74	14	71	68	39
Race	1 hr. after	85	20	—	95	65
	4 hr. after	85 (+15%)	44 (+214%)	82 (+15%)	88 (+29%)	68 (+74%)
6/23	24 hr. after	92	54	106	133	142

*Expressed in eosinophils per cu. mm. of blood.
†Had hay fever; had undergone splenectomy five years previously.
‡Had hay fever.

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distance from the city, submitting to a strict and regular program, including two daily rowing periods, excellent diet, ample rest and sleep, very little physical exercise other than rowing, and minimal exposure to extraneous emotional or other forms of stress. Time trials over the regular course were made on the twelfth, eighth and sixth days before the race. Since the times obtained were nearly all identical under essentially similar conditions, comparable muscular-energy output may be postulated.

Blood samples were obtained at intervals of one or two days, either immediately before the morning rowing period or immediately before the evening period. For the races and for selected time trials blood samples were obtained immediately before leaving the boathouse, immediately on returning, within one hour after race or time trial, and four hours and twenty-four hours after the beginning of the race.

For this study two crews were selected: the Harvard varsity crew (ages 19 to 24, weights 177 to 193 lb.), the eosinophil response being studied after the first and third time trials and after the Harvard–Yale race, and the Harvard combination crew (ages 17 to 22, weights averaging 175 lb.), the eosinophil response being studied after the second time trial and after the race. The varsity crew raced four miles, the combination crew two miles.

All oarsmen studied had been carefully examined by members of the staff of the Harvard University Department of Hygiene at the beginning of the season. Further medical histories were obtained from each, with particular attention to any past or family history of allergy. Physical examinations were performed on special indication only. During and after training, time trials and races both crews were closely observed and the evaluation of individual effort, fatigue and stress was attempted.

Capillary blood samples were obtained from the ear lobes. The eosinophil-counting technic described by Randolph[5] and modified by Henneman and others[6] was used. Levy counting chambers of 0.2-mm. depth were used, and 4 entire chambers were counted each time. This method, with Phloxin B in propylene glycol and water as staining fluid, proved to be well adapted to field studies, since preliminary examination revealed no lysis of eosinophils within twenty-four hours. However, counts were made within six hours after blood withdrawal.

TABLE 1 (*Concluded*).

DATE	BLOOD SAMPLED	No. 6†	No. 7	No. 8	COXSWAIN	COACH
6/9	Control	90	48	78	12	
6/10	Control	114	110	56	114	
6/11	Just before	68	99	72	24	
First time trial	1 hr. after	82	92	49	28	
	4 hr. after	21 (−68%)	16 (−84%)	12 (−83%)	24 (−0%)	
6/12	24 hr. after	183	180	114	92	
6/14	Control	136	91	79	68	
6/16	Control					
6/17	Just before	107	149	82	98	
Second time trial	1 hr. after	110	82	56	58 (−40%)	
	4 hr. after	28 (−74%)	42 (−72%)	32 (−60%)	91	
6/18	24 hr. after	86	107	86	104	
6/20	Control	90	95	54	48	
6/22	Control	136	112	62	17	
6/23	Just before	24	44	23	33	101
Race	1 hr. after	17	27	14	3	6 (−94%)
	4 hr. after	14 (−42%)	3 (−93%)	3 (−88%)	3 (−91%)	17
6/24	24 hr. after	72	68	65	115	177

TABLE 2 (*Concluded*).

DATE	BLOOD SAMPLED	No. 6	No. 7‡	No. 8	COXSWAIN‡
6/15	Just before	98	316	96	369
Time trial	1 hr. after	74	242	37	330
	4 hr. after	30 (−69%)	224 (−26%)	31 (−68%)	384 (−6%)
6/16	24 hr. after	157	262	60	328
6/20	Control	39	154	82	204
6/22	Just before	34	264	27	33
Race	1 hr. after	66 (+94%)	156	27	95 (+188%)
	4 hr. after	41	101 (−61%)	24 (−11%)	—
6/23	24 hr. after	98	237	32	237

Results

The results are summarized in Tables 1 and 2 and Figures 1 and 2. Certain observations are of interest.

The control eosinophil levels of each subject, as taken before rowing, showed a definite tendency to stabilize within a certain range. With the exception of allergic persons, this range was rather low, varying between 65 and 168 eosinophils per cu. mm., with an average of 108.

In the varsity crew, within four hours of the first time trial the original average eosinophil level of 123 was reduced to 19 and after the second time trial to 38. Immediately before the actual race the average count was only 64 and fell to 3 within four hours thereafter. It is of interest that in certain subjects the lowest level of circulating eosinophils was reached after one hour, whereas in the majority it was reached only after four hours. It should be emphasized that the varsity four-mile race was extremely hard and tense, won by only a quarter of a length, with a spectacular and (to the crew) frightening finish.

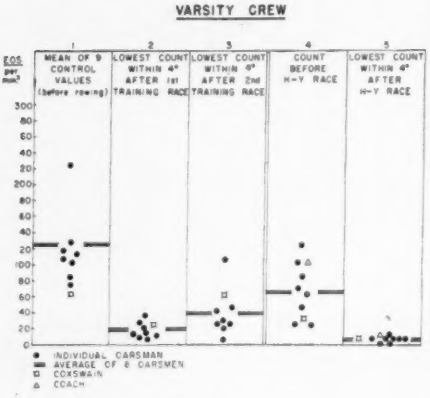

FIGURE 1.

In the combination crew, adjustments had to be made for the high eosinophil counts of two oarsmen with active hay fever. After the time trial the average of the remaining six oarsmen fell from an initial count of 108 to a final 56. On the day of the actual race the initial count was low, averaging 42, and there was no fall at either one or four hours; indeed, there was a slight mean rise to 59 eosinophils per cu. mm. The race differed greatly from the varsity race, since it was won by six lengths, with Harvard leading easily all through and with no doubt of the outcome. Before the race, however, there was considerable emotional tension, since this was the first race in the regatta.

On race day coxswains and coach showed an eosinophil response comparable to the oarsmen's, whereas during the training period this was not the case.

Eosinophil counts at twenty-four hours, obtained primarily to ascertain whether abnormally high eosinophil levels might then be present, were found to be within the initial range.

Six men with a history of allergy showed high initial eosinophil levels but otherwise responded in the same fashion as the other oarsmen. It is of interest that the eosinophil count of the coxswain of the combination crew dropped on the day of the race from an average of 300 or more per cu. mm. to 31 before the race, then rose to 94 after the "easy" race itself. Furthermore, on several occasions the men with active hay fever described a definite subjective improvement for several hours after either a strenuous rowing period or a time trial.

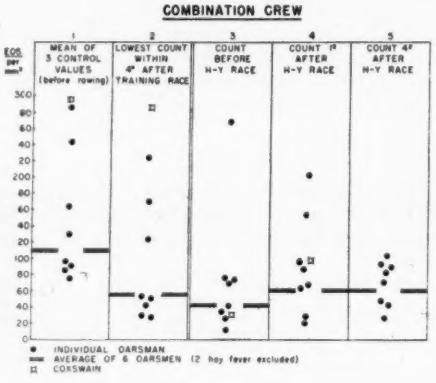

FIGURE 2.

One member of each crew (No. 5 of the varsity crew and No. 3 of the combination crew) had undergone splenectomy four and five years previously, after traumatic rupture of the spleen. Both had in addition a past history of allergy. Neither showed any deviation from normal eosinophil response.

The impossibility of satisfactory evaluation of personalities precludes more than an overall impression of the relation of personality types to the reaction patterns observed. Low control counts were obtained in tense subjects with a tendency to feel increased anxiety and sense of responsibility. The coxswain of the varsity crew was an example of this type, carrying the responsibility of being both

coxswain and captain. Control counts as low as 24 and 17 were obtained. On the other hand, No. 5 in the combination crew, the only oarsman whose circulating eosinophils increased after the time trial, appeared definitely phlegmatic; moreover, being the strongest member of the crew, he did not need to drive himself to exhaustion.

Discussion

If a decrease in the level of circulating eosinophils under these conditions may be considered a valid index of an increase in circulating adrenal oxysteroids,[1] one may well conclude that stimulation of the adrenal cortex does occur during competitive strenuous physical exercise. Two major factors are involved: intense muscular activity and emotional stress. From the data here presented, the great importance of emotional factors clearly arises. Emotional stress, either of itself (coach, coxswains, initial count on race day) or in combination with muscular activity (varsity crew race), represents a maximal stimulus for the stress mechanism studied, whereas this does not seem to be true of muscular activity alone. This, of course, relates to well trained persons only. The evidence presented would, therefore, tend to establish more securely the importance of the cerebral cortex in the existing schemata of the stress reaction.[4]

A practical application suggests itself: In unselected groups eosinophil counts made under conditions of standardized stress may well facilitate the identification of persons with unsatisfactory adrenal response to stress and persons physiologically well prepared in whom the particular exertion does not exhaust physical or emotional reserves. Both groups will show apparently inadequate eosinophil response, but the clinical difference between the two will be obvious at a glance. The simplicity of the procedure would permit its use as a screening device.

Summary

Adrenal cortical response to standard physical exertion and added emotional stress, as measured by changes in circulating eosinophil levels, was studied in healthy, well trained oarsmen. Members of the Harvard varsity and combination crews were observed during a two-week training period and immediately before and after contests with Yale crews.

Significant changes were easily demonstrated. From the evidence obtained it was concluded that in well trained persons emotional stress, either alone or in combination with muscular activity, may lead to a highly effective adrenal stimulation and may represent a necessary link in the mechanism of adaptation.

Practical application of the simple technic as a screening device for the estimation of adrenal function under various conditions was suggested.

In men with active allergy and in two subjects whose spleens had previously been removed no deviation from normal response could be detected.

We wish to express our appreciation of the good-humored co-operation of the Harvard crews and their head coach, Mr. Thomas D. Bolles.

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GENITAL DERMATITIS CAUSED BY PENICILLIN: ITS RESPONSE TO NICOTINIC ACID*

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PELLAGROUS dermatitis of the genitals has been recognized for years. Redness, swelling, vesiculation, fissures, scaling and even bullae may occur. Vaginitis, vulvovaginitis, proctitis and scrotal and inguinal inflammations have been described. This type of pellagra can (and commonly does) occur without concomitant dermatitis of the areas usually involved in this disease — the parts exposed to the sun, the mouth and so forth.

In the past five years, 20 cases of genital and rectal dermatitis have been observed to result from the administration of penicillin. All 20 cases have responded dramatically to the administration of nicotinic acid by mouth and cold saline compresses applied locally. There can be no question that the administration of penicillin precipitated this pellagrous genital and rectal dermatitis, but its biochemical cause can only be speculated on.

FIGURE 1. *Erythema, Papules, Scales and Crusts.*

For centuries the etiology of pellagra was in dispute, and it was not until 1924 that Goldberger[1] showed that dried brewer's yeast benefited pellagrins. In 1937 it was found that nicotinic acid cured most cases.[2, 3] Since not all pellagrins responded to nicotinic acid, many attempts have been made to find the cause. Intestinal micro-organisms are known to convert tryptophane to nicotinic acid. The evidence available at present[4] indicates conclusively that mammalian tissue also can convert tryptophane to nicotinic acid.

Recent studies[5] also have demonstrated that other amino acids, especially some found in corn, seem able to inhibit the formation of nicotinic acid from tryptophane, as persons fed on a diet rich in corn develop pellagra even if the diet contains niacin in amounts that are ordinarily sufficient to afford protection. In this regard it is interesting to note that early writers on dermatology — Duhring in 1886,[6] Hyde in 1900[7] and Jackson in 1901[8] recognized the importance of corn as an etiologic factor in this disease. Hyde[7] stated: "Pellagra originates in the use as an article of diet of maize (corn) which has been improperly dried or has developed deleterious substances after its reduction to a coarse powder." Lambroso (quoted by Hyde) in 1898 experimentally produced the symptoms of the disease from "toxines" developed during the spoiling of maize (corn).

These pioneer dermatologists recognized in the 1880's what biochemists of today are proving to be true: that is, that corn contains some antifactor or factors that can retard the conversion of tryptophane to nicotinic acid.

It has recently been shown that penicillin, a complex monobasic organic acid, can inhibit peptide synthesis in lower animals. Such interference was largely attributed by the authors to its interference with the utilization of the amino-acid glycine of an amino-acid mixture used for the growth of a bacterial strain.[9]

It is not too difficult, then, to theorize that penicillin in man may be able to interfere with the utilization of the amino-acid tryptophane or prevent its conversion into niacin, or both. Experimental work along this line should be undertaken, for we know today that the "simple" condition of pellagra is not so "simple" but is a most complex entity. Several other factors will probably be found to make the disease even more complicated than it seems today.

The histories of the 20 cases observed have been generally similar; that is, during or immediately after a course of treatment with penicillin the patients developed redness, vesicles, papules, moisture and scaling of the genital or rectal areas or both (Fig. 1). Five of the 20 cases had diarrhea, and 4 had stomatitis. Not one had the areas of pellagrous dermatitis that usually result from exposure to sunlight. Most of the cases were seen in patients who had been given penicillin after an operation.

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No correlation of environmental, racial, financial or dietary factors in the patients' histories was observed. The 20 patients were seen in consultation and were closely followed by the referring physician and the author. No local or internal medications were used, other than nicotinic acid by mouth (100 mg. 3 times daily) and cold saline compresses on the affected parts. Healing was usually complete within twelve days of institution of such therapy. The majority of patients had never previously received penicillin.

Case Report

W. B., a 40-year-old man, was injured at work and underwent an operation at a hospital. On the second day of penicillin therapy he noted itching, redness and "little whiteheads" on the scrotum and the sides of the thighs. He was given local treatments while in the hospital and was then referred to his own physician, who continued treatment. The rash failed to improve, and he was referred for consultation. On examination the scrotum was red, moist and eczematous and also showed crusts and scaling. There were scattered papules on the inner legs, extending downward from the groin. A diagnosis of pellagra resulting from penicillin therapy was made. The patient was given nicotinic acid, 100 mg. 3 times daily, with instructions to apply cold saline compresses locally. The patient was discharged in fourteen days, with the eruption completely cleared.

Summary

Twenty cases of scrotal and rectal pellagra have been observed to follow routine penicillin therapy.

Nicotinic-acid metabolism is probably deranged by penicillin.

Nicotinic acid should be given immediately to patients who have any scrotal or rectal itching or redness during or after penicillin therapy.

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FIFTY YEARS OF MEDICAL PROGRESS

MEDICINE AS A SCIENCE: PSYCHIATRY

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PSYCHIATRY is regarded by many as the youngest among the medical sciences and by some is considered not "scientific" in many of its aspects. It is, without question, a highly controversial field of medicine, yet the last fifty years have witnessed its phenomenal growth as one of the recognized medical disciplines, and it has exerted a tremendous influence that reaches far beyond the confines of medicine and has become the object of both *belles lettres* and the cartoon. How much of this large field will survive for another fifty years and in what manner it will continue to manifest itself it is difficult to predict, but even more urgently than prediction is needed an ever greater effort to replace opinion and speculation with a wide body of proved facts and secure knowledge.

When one undertakes to view it in historical perspective, it now seems that the basic observations on which the whole development of psychiatry during the last fifty years was founded were available or just about to be made at the turn of the century. Noguchi[1] had finally demonstrated the special pathology of general paresis and had revealed *Spirochaeta pallida* as the etiologic factor in this hitherto perplexing disease and its multiform manifestations. For a time brain histology and new staining methods for laboratory investigations seemed destined to open a sure path for the understanding of almost all psychoses. Subsequently, Alzheimer's disease,[2] senile psychoses, Huntington's chorea and mental deficiency all were revealed as behavior abnormalities related to impairment of brain function; but histopathology has failed to give a definitive answer for schizophrenia and related disturbances.

Psychoses are now regarded not only as disturbances of rational thought but also as related to emotional factors that in turn control thinking, reasoning and judgment. The nature of *emotional* responses was destined also to be better understood after the turn of the century, and new and crucial insights were attained. It was at that time that Cannon[3, 4] began his studies of gastrointestinal motility in response to the presence or absence of adrenalin; his findings proved to have a significance that far surpassed his expectation or the area of his initial concern. Emotions then began to appear as

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physiologic events with psychologic aspects, controlled by chemical factors and related to the individual's adjustment to the demands and opportunities presented him in the outer world. Demonstrations of the hypothalamus as the co-ordinating center for emotional expression and a whole series of observations in comparative anatomy that have recently been enumerated by Cobb[5, 6] have served to place reasonably unchallengeable knowledge about the physiologic component of emotions at the disposal of the psychiatrist.

FIGURE 1. *Sigmund Freud.*

Pavlov's discovery[7-10] that emotional reactions and the concomitant visceral activity were related to the pressure of specific stimuli and that the responses could be conditioned by proper timing of the stimuli formed the basis of a tremendous development in the understanding of the processes of learning in both the lower animals and man. The basic concepts, which were elaborated by Watson[11] in behavioristic terms and by Clark Hull[12] in a systematic learning theory, have proved to be good tools for studying the phenomena of neuroses under the hypothesis that they are results of faulty learning.

The investigation of the effect of past experience on subsequent behavior, which has been described by some as "the historical approach to the individual," was also based on work done at the turn of the century. At that time Sigmund Freud had just published his joint observations with Breuer,[13] and he was then engaged in the study of dreams as an avenue to the understanding of neurotic behavior conceived as the consequence of traumatic experience.

The last five decades have seen an elaboration of these concepts into impressive theoretical structures and the accumulation of a large body of laboratory and clinical observations, all of which essentially still rest on the four cornerstones of brain pathology, physiology of the emotions, the learning theory and the psychoanalytic study of the unconscious. These fifty years have also seen the advancement of psychiatry into the field of social reform. The nineteenth century had been a period in which moral recrimination directed toward the mentally ill was replaced by an attitude of objective study and an emphasis on humane treatment. During the century much effort was expended in efforts to improve the care of the mentally ill and to bring before the general public those insights about mental disease that had been more or less securely established. The mental hygiene movement that started in 1908[14, 15] is still vigorous and has continued to direct the attention of research workers to various phases of early childhood development, in the hope of making possible the prevention of later mental or emotional breakdown by early detection of cases and early treatment. There have also been worthwhile applications of psychiatric concepts to penology, although the borderline between delinquency and crime, on the one hand, and deviant behavior interpreted as illness, on the other, has not even now been satisfactorily determined.

Psychiatry holds a curious position in scientific thinking. On the one hand it is a field in which objective understanding of brain functions and the manipulation of physiologic systems by means of surgery, drug therapy or electrical stimuli is regarded as the proper avenue of approach; on the other hand, it is an area in which there is also an attempt to understand the sick person in terms of his life experience, his motivations and goals and the obstacles he has met in his attempts to secure satisfaction for his basic needs. Brain anatomy, physiology and biochemistry, — especially the chemistry of the hormones, — have provided advance along the one road, while psychoanalytic theory and technics and the development of various schools of psychotherapy have facilitated progress along the other.

One may say that as of 1950 both biologic psychiatry and social psychiatry have made large strides toward the understanding of the field of mental illness, but until now these have not been well co-ordinated; the great task of the future would seem to be greater efforts to integrate the two fields. The most promising milestones so far in this en-

deavor seem to be the discoveries of recent years, particularly the new insights made possible by brain surgery in the frontal lobe,[16] the application of a variety of drugs to alter mental functioning, such as sodium amytal[17-20] and mescaline,[21] the discovery of hormones clearly related to changes in mood and motivation,[22] and formulations co-ordinating biologic and psychologic phenomena, such as those provided by the concept of the general adaptation syndrome.[23] Furthermore, it is only during the last ten years that we have seen the application of rigorous and classic laboratory methods to the study of states of feeling and to emotional attitudes as they are related to changes in visceral activity. Harold Wolff's monumental work[24] in bringing together what is now known about the relation of life stress to somatic disease is a landmark in pointing out that psychiatry has at last found a secure place in the general hospital and in the fields of internal medicine and surgery.

The century began with a psychiatry concerned with diseases of special organs and with the invasion of the human organism by specific agents. Freud and Cannon set the stage for efforts to describe and understand the organism as a whole in terms of its behavior, organization and adjustment. Adolph Meyer,[25, 26] founding the movement of so-called psychobiology and describing the individual human being as both product and victim of his own life experiences, began reviewing the complaints of psychotic and neurotic individuals in the light of their assets and liabilities in the process of adjustment to the tasks provided by their human environment. The study of intellectual equipment, motor and social skills and handicaps due to defects in equipment came to be of concern to the psychiatrist — and much help was needed from the psychological laboratory.

At the turn of the century Kraepelin[27] had become interested in testing intellectual abilities according to the methods then carried out in Wundt's laboratory. Ebbinghaus had already tried to develop standard tests for reading and memory. Binet and Simon[28] finally developed standard procedures for the assessment of intellectual equipment at different age levels and for the appraisal of intellectual deterioration in acquired mental disease. Out of the efforts of these workers developed the movement of clinical psychology, which in the last decade has reached the status of a separate profession working hand in hand with the psychiatrist and making its contribution in the fields of measurement of mental functions and assessment of changes in emotional attitudes by projective technics. Since the second world war there has even been a strong movement toward participation by the clinical psychologist in psychotherapy as well as an enlargement of his scope of activity, so that he now may take over large sections of the psychotherapeutic efforts of a psychiatric clinic. The psychologist is now indispensable in the careful study of the effects of drugs and surgical procedures on the brain, and psychological testing will continue to develop as psychiatry expands its most recent area of concern, the field of preventive psychiatry.

The screening of whole populations for the presence of vulnerable conditions in personalities and the planning of preventive measures at the community level require testing programs that can be aligned with physical examinations.[29, 30]

As a member of a so-called auxiliary profession, however, the clinical psychologist still cannot provide all the additional information needed in the more modern approaches. The human being is still not fully explained as a whole until he is seen as a member of a group and as enmeshed in family or friendship groups, work teams and social institutions. The practical task of rehabilitation of veterans of the first world war required the cooperation of the emerging profession of social work. Women trained in this field were able to lend a hand to the person seeking to master situations of social stress. Although at first it seemed that poverty, job trouble and poor living conditions might be blamed, it soon became evident that the complex variety of roles that the individual must fill as child, parent, worker and citizen in a cultural setting constitute the matrix in which adaptations must take place and that only scientific understanding of this matrix will make it possible to answer some of the basic questions of psychiatry.

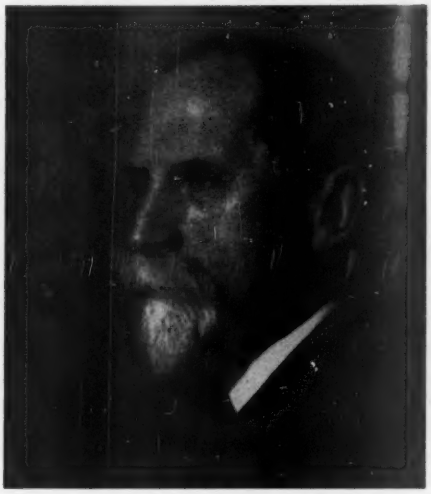

FIGURE 2. *Adolf Meyer.* (*Reproduced by Permission.*[26])

This recent development has brought psychiatry into close contact with social science. Efforts are being made to develop interdisciplinary research under the auspices of such groups as the Institute of Human Relations at Yale, the Commission of Human Development at the University of Chicago and the Department of Social Relations at Harvard. Comparative anthropology begins to provide some information about the kinds of emotional and mental disturbances seen in different cultural groups.[31, 32] Sociologic analysis of roles at various levels of society and of the factors that guarantee the continuation of a functioning society irrespective of the specific individuals living in it[33] and, finally, the study of group dynamics by social psychologists[34] are all new fields of endeavor to which psychiatric clinical knowledge contributes and from which, in turn, it receives new tools for the solution of its own problems.

It seems that we are standing at the beginning of a large new development in the study of man that will be built on the integration of the efforts that have been made in the last fifty years in a number of fields and to which psychiatry will make a major contribution.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

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CASE 37201

PRESENTATION OF CASE

A forty-year-old woman entered the hospital because of increasing weakness for four weeks.

Four and a half months before admission a hemoglobin of 12 gm. per 100 cc. was discovered. After she had taken liver extract and iron over the course of the next month, the hemoglobin was unchanged and blood studies showed a microcytic hypochromic anemia, with a reticulocyte count of 1.1 per cent. Iron was given but was discontinued after 25 capsules had been taken, because of constipation. During this time there was increasing anorexia, a poor diet and an 8-pound weight loss. Four weeks before entry an attack of "grippe" occurred, with rapid onset of weakness, muscular aches, chills, a high fever, a brief period of delirium, and pain in the epigastrium and right hypochondrium, which was almost constant and persisted up to admission. After one week in bed the patient returned to her work as a secretary, feeling tired, weak and dizzy and noticing dyspnea and palpitation on exertion and night sweats on 5 occasions. There was no history of melena, hematemesis or jaundice.

The past history was one of good health, with no significant illnesses. In the six or seven months after entry her previously normal periods had become very scanty but occurred regularly.

Physical examination revealed a thin, very pale woman. Firm bilateral slightly enlarged axillary lymph nodes and a left supraclavicular lymph node were barely palpable. There was slight right-upper-quadrant abdominal tenderness and voluntary spasm. The liver and spleen were not felt. The chest, pelvic and rectal examinations were not remarkable.

The temperature was 100°F., the pulse 110 and the respirations 20. The blood pressure was 110 systolic, 74 diastolic.

Examination of the blood revealed a hemoglobin of 6.25 gm. per 100 cc. and a hematocrit of 24 per cent; the red cells were microcytic and hypochromic. One reticulocyte count was reported as 0.6 per cent and a white-cell count as 9000, with a normal differential and increased platelets. Bleeding time was 4½ minutes, clotting time 8 minutes. The prothrombin time was 24 seconds (normal, 19 seconds). The van den Bergh reaction was less than 0.4 mg., the serum iron 193 microgm. (normal), the total protein 6.75 gm., the albumin 3.84 gm., the globulin 2.91 gm. (albumin-globulin ratio 1.3), the nonprotein nitrogen 21 mg., the uric acid 2.6 mg. and the fasting blood sugar 96 mg. per 100 cc. The urine was normal. Three stool specimens were guaiac negative; one was guaiac + to ++. A urobilinogen test showed a normal excretion. Agglutinations for typhoid and undulant fever were negative. A smear of bone marrow from the iliac crest was initially interpreted as showing erythroid depression and relative myeloid hyperplasia. A tuberculin test in a dilution of 1:100,000 was negative; 1:10,000 was + in forty-eight hours. X-ray films of the chest, barium-enema examination, gastrointestinal series and bone survey were negative. A hematologic consultant found that the reticulocyte count was 5 per cent and interpreted the bone-marrow aspiration as intense erythroid hyperplasia with orderly maturation in the myeloid series. A small-bowel enema given with a Rehfuss

FIGURE 1. *Film from Small-Bowel Study.*
Arrows point to two areas of involvement of the jejunum. The third area is not clearly seen on this film.

tube revealed that barium flowed readily through the distal duodenum and proximal 1 ft. of jejunum into a loop of jejunum in the right upper quadrant, where it encountered a mass protruding into the lumen of the small bowel and widening the small bowel. A normal mucosal pattern was not seen in this area, and the lesion appeared to be extensively ulcerated. The head of the column was lost while this area was being studied, but follow-up films showed two lesions, one in the right upper quadrant and the other in the left midabdomen. Thus it could not be determined whether there were two or three lesions in the small bowel (Fig. 1).

On the seventeenth hospital day an operation was performed.

Differential Diagnosis

Dr. Chester M. Jones*: It seems to me that certain comments can be made about this history and certain guesses can be made about the diagnosis. The story that the patient had had increased weakness for only four weeks I think is probably not accurate, because I do not believe the blood would have been examined four months before entry unless she was not feeling well. Certainly a hemoglobin of 12 gm. is not particularly low. The fact remains that she did have a hemoglobin of 12 gm. and that four months later it was 6.25, which gives one a definite idea of the amount of blood loss or destruction taking place. The blood smear showed microcytic hypochromic red cells, and that suggests actual blood loss. Blood loss could be due to excessive menstruation, but her periods, although regular, were scanty, so she had lost less than the usual amount from that source.

Then there was an attack that was called grippe, which had all the earmarks of an acute infection — malaise, weakness, chills and a temperature high enough to cause the patient to become somewhat disoriented. In addition she had almost constant pain in the epigastrium and hypochondrium that persisted up to the time of admission. That is curious. The pain was not peristaltic and was not associated with other symptoms of intestinal obstruction, such as nausea and vomiting. She had the symptoms of increasing anemia — dizziness, weakness, fatigue and dyspnea and palpitations on exertion. The white-cell count suggests but does not prove that she had an infectious process. It could also have been due to the extreme weakness or to more bleeding than just a steady oozing. The fact that there was no history of melena does not prove that there was or was not bleeding from the gastrointestinal tract. I think I can accept the story of no hematemesis and no jaundice, but I am not at all sure that she ever examined her stools carefully to see if they were dark or not. I should like to point out that no bowel symptoms had apparently been noted, such as a change in the shape or frequency of the stools, except for constipation during the time she was receiving iron.

On physical examination she merely showed pallor, loss of weight due to impaired appetite and palpable lymph nodes in both axillas and in the left supraclavicular area. She had lost weight and was very thin, so it is possible that the lymph nodes were of no significance. However, the history does say that the left supraclavicular lymph nodes were enlarged, so I cannot entirely forget them. Perhaps they have some bearing on the attack of fever and muscular pains a month before entry. Could that have been infectious mononucleosis with a residual lymphadenopathy? Although that is possible, the laboratory findings do not suggest it, so I am forced to discard that explanation. I have always considered that an enlarged left supraclavicular lymph node might mean carcinoma of the stomach, but it may also be seen with other neoplastic disease in the upper abdomen; therefore I should seriously consider tumor here.

The laboratory findings were remarkably negative except for the evidence of a hypochromic type of anemia. The hematocrit was 24, and there was a flat statement that the red cells were microcytic and hypochromic. The reticulocyte count of 0.6 per cent does not indicate a good response to the anemia. I think the normal serum iron is a common finding in an iron-deficiency type of anemia, whereas in pernicious anemia or hemolytic anemia the serum iron is increased. Of four stools, one was + to ++. Ordinarily I should not pay too much attention to a + guaiac test, but I cannot overlook a ++ guaiac. I believe she was bleeding from the gastrointestinal tract, and I am sure the physicians studied her with that in mind as the most likely source of the anemia. Among other things, two bone-marrow biopsies were done. I think I have a right to accept as correct the interpretation of the second, which evidently was studied very carefully. It seems to me that the response of the bone marrow, namely intense erythroid hyperplasia with orderly maturation of the myeloid series, rules out leukemia and a myelophthisic type of anemia. The platelets were not diminished. This does not seem to be the picture of tumor encroaching on the bone marrow but rather a response to a profound anemia from blood loss.

Routine x-ray films were taken. Then, because they were normal, because the patient was probably bleeding from the gastrointestinal tract and because small-bowel disease may be quite silent, special x-ray films were taken. May I see the films, Dr. Wyman?

The spleen looks normal in size; that is of some comfort when one thinks about a lymphoma. The stomach is normal and the large bowel discloses nothing contributory.

Dr. Stanley M. Wyman: It is incorrect that only routine films were done during the first examina-

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tion, Dr. Jones. The film from the barium-enema examination demonstrates no intrinsic disease. As Dr. Jones pointed out, the spleen does not appear enlarged and the liver is not remarkable. The x-ray films of the upper gastrointestinal tract show a small hiatus hernia and a grossly normal-appearing stomach, duodenal cap and loop. At that time the examiner believed he ought to look at the small bowel, so the patient was given ice water and the small bowel was examined as thoroughly as possible. Unfortunately, there was still barium in the colon. One lesion of the small bowel was detected at the time that this was done. I cannot suggest any second or third lesion on this examination. The examination of the small bowel done with the Rehfuss tube discloses a very different picture. As an examiner followed the barium from the ligament of Treitz into the jejunum, the barium suddenly struck a lesion, which he recorded on spot films. Although the barium column had been stopped, while the examiner was taking the films it moved far enough ahead that he lost track of it. When he next looked at the head of the column he saw a second lesion, and finally what he thought to be a third lesion. He was sure that there were two lesions but he could not be sure of the third (Fig. 1).

Dr. Jones: By the technic of rapid filling of the small bowel under fluoroscopic observation and the taking of spot films, two and probably three lesions have been shown beyond the ligament of Treitz. It seems to me they probably are dilated loops of small bowel above a narrowed portion, with involvement of the entire mucous membrane. It looks to me more like an inflammatory process than a tumor pushing into a loop of small bowel. I should like your opinion on it, Dr. Wyman.

Dr. Wyman: I interpret it differently, Dr. Jones. I think it is a mass pressing into the lumen of the bowel and destroying the mucosa. The two or three lesions all look the same. I think it is probably tumor.

Dr. Jones: There was focal disease in the small bowel in two or three places, and possibly in other places that could not be visualized. What could produce this sort of lesion and also produce an iron-deficiency anemia? The anemia can be explained on the basis of blood loss that was not obvious. Then one considers the various tumors and inflammatory processes that involve the small bowel. Of the inflammatory processes, I think the only one proper to mention is regional enteritis. A very few cases have been seen with patchy involvement of the ileum and further involvement progressing toward the pylorus. Regional enteritis usually shows narrowing with dilatation above the narrowing but no evidence of tumor formation. In view of Dr. Wyman's interpretation, in which I place a good deal of confidence, I cannot explain the lesions on the basis of regional enteritis.

In discussing the possibility of tumor, I do not think it is worth while to list the entire group of neoplasms that may occur in the small bowel. Benign tumors of the small bowel are said to occur in about one per cent of autopsy specimens. They are not common, but they are not extremely uncommon. In the order of frequency, they are: adenomas, leiomyomas, pancreatic rests, lipomas, fibromas, hemangiomas and so on. Of the benign tumors, a certain number eventually become malignant. The adenomas and the myomas become malignant in possibly eight to ten per cent of the cases, but it is very difficult to find an accurate figure.

Among the frankly malignant tumors — I think they are really less common than benign tumors — is adenocarcinoma. Adenocarcinomas, according to statistics, are not frequently found in the duodenum proximal to the ligament of Treitz or in the ileum. Although they can occur anywhere, they are also rather uncommon in the jejunum. Myosarcoma, a malignant type of myoma, can occur in various parts of the small bowel, but the jejunum frequently seems to be spared. What else could it have been? It is well known that lymphoblastoma produces spotty lesions in the gastrointestinal tract, but the stomach and ileum are the two most common places. In the case under discussion the disease was in the jejunum and in the ileum but not in the terminal ileum. In about two thirds of the cases of lymphoblastoma that have been described adenopathy was noted. I should guess that a fair number had enlargement of the spleen. In this case the spleen did not appear to be enlarged clinically or in the x-ray films, and there was no gross generalized adenopathy. I am still interested in the axillary and supraclavicular lymph nodes, and I think a biopsy would give the answer. However, they may have been lymph nodes that were easily felt in a person who was very much underweight. Finally, a lymphosarcomatous process that had produced this degree of anemia would have produced more evidence of disease.

Although the tuberculin test was positive at 1:10,000, I do not believe she had tuberculosis, nor do I think she had syphilis. I should rule out an unusual type of regional enteritis on the basis of Dr. Wyman's interpretation of the x-ray films. I think she had a neoplastic process, — by the law of chance, a leiomyoma or an adenocarcinoma, — with several separate tumors. I believe the anemia was posthemorrhagic. Obviously the surgeons should have operated on this patient to make a diagnosis and to determine whether or not the disease could be removed.

Dr. Perry Culver: I should like to make several comments. The patient was first seen by a dermatologist, who wanted to give her sulfadiazine because of an infection of the left heel. At that time the hemoglobin was 12 gm. Then someone gave her

iron and liver. I do not know whether or not she received the sulfadiazine. When I saw her the night before admission she stated emphatically that the symptoms of burning, sour stomach and vague tenderness in the epigastrium had been present for only one to two weeks. Because of the hemoglobin of 6.25 gm. and the fact that she gave a history of vague upper-abdominal symptoms, I thought she ought to be admitted to the hospital for study.

Dr. Jacob Lerman: This patient was on my service. Because she had a blood-loss type of anemia we studied her along the lines indicated in the history. The blood and x-ray examinations were all reported negative. At first I placed some

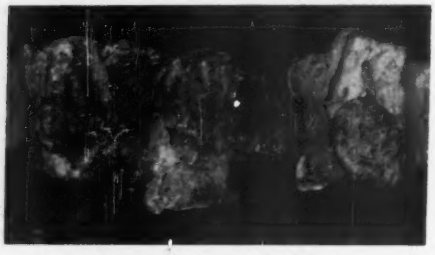

Figure 2. *Resected Small Bowel.*

emphasis on the hiatus hernia in a young person as a source of chronic blood loss but later discarded it. When we received the first report of the bone-marrow studies, we considered the possibility of aleukemia or agnogenic myeloid metaplasia. After Dr. Jacobson examined the bone marrow, we decided to repeat the x-ray examinations. It was then that the small-bowel series was done.

Clinical Diagnosis

Lymphoma of jejunum.

Dr. Jones' Diagnosis

Tumor of small bowel; ?leiomyoma, ?adenocarcinoma.

Anatomical Diagnosis

Malignant lymphoma, Hodgkin's sarcoma type, of jejunum, multiple.

Pathological Discussion

Dr. Tracy B. Mallory: At operation a group of three separate tumors was found localized to a segment of bowel about 25 cm. in length. The mesenteric lymph nodes were enlarged but soft and obviously nonneoplastic. In the picture of the resected bowel the lesions are annular and ulcerated (Fig. 2). The bulk of the tumor appeared to be in the submucosa, with relatively little involvement of the mucosa but with extension out through the muscle layer to the serosa. Histologically, it was obvious that the tumors belonged in the lymphomatous group, but as is so often the case, the experts are likely to disagree about the exact name that should be given to them. In many laboratories the pathologists would undoubtedly call them reticulum-cell sarcomas. I think most of the pathologists in Boston would call them Hodgkin's sarcomas. Quite commonly tumors are seen that appear to be in a transitional stage between these two types. A number of mesenteric lymph nodes were examined and were free from involvement. As yet no biopsy of the axillary or supraclavicular lymph nodes has been done, so we still do not know whether the tumor was limited to the bowel or was generalized.

Dr. Jones: This is very important. Lymphoma that is sharply limited to the gastrointestinal tract can be removed with excellent results; with adenopathy in addition, the outlook is quite different.

Dr. Mallory: It would also be very important to examine the lymph nodes to determine whether to treat the patient further with x-ray or possibly with chemotherapeutic agents.

Dr. Jones: Why was a biopsy not done? There may have been a very good reason.

Dr. Lerman: The "enlarged" lymph nodes did not impress us as being abnormal.

Dr. Jones: The patient was malnourished, so normal lymph nodes were palpable!

Dr. Culver: How frequently do lesions like this ulcerate and bleed?

Dr. Mallory: It is fairly common. Any malignant tumor is likely to do that.

Addendum

The patient left the hospital about ten days after operation but returned six weeks later because of weakness, back pain and vomiting. A chest x-ray film showed a lesion in the right upper lobe, for which she received x-ray treatment of 1200 r. She was markedly anemic, and bone-marrow aspiration studies showed neoplastic involvement. She was given a course of ACTH (adrenocorticotrophic hormone) without improvement and recently (three months postoperatively) left the hospital against advice.

CASE 37202

Presentation of Case

A sixty-two-year-old man was admitted from another hospital because of massive intestinal bleeding.

The patient was well until two months prior to admission, when he developed vague gnawing upper-abdominal distress. One month later a physician prescribed nicotinic acid for dizziness of four to

five days' duration. In the next five days the patient became increasingly weak and three weeks before entry was admitted to another hospital after passing tarry stools and collapsing at work. On admission there his temperature and pulse were normal. The blood pressure was 110 systolic, 80 diastolic. The patient appeared somewhat pale but was not in shock. There was slight tenderness in the lower part of the midepigastrium, but no spasm and no masses could be felt. Rectal examination revealed tarry stool on the examining finger. The hemoglobin was 11 gm., the red-cell count 3,400,000 and the white-cell count 7500, with a normal differential. On the following day the hemoglobin had fallen to 7.5 gm. and the red-cell count to 2,600,000. The prothrombin time was 100 per cent of normal. Two days after admission a barium-enema examination showed filling of the colon and the appendix with a large amount of fecal residue; no defect was demonstrable. The patient required approximately 1000 cc. of blood daily to maintain the hemoglobin at between 7 and 8.5 gm. He was nourished by means of hourly feedings of milk and cream. Gastric aspiration on the third day revealed gastric juice and milk curds but no evidence of blood. On the fourth hospital day a gastrointestinal series demonstrated a small hiatus hernia but no lesions in the esophagus, stomach or duodenum. A chest film showed mottled densities in both lung fields, extending from the hilum to the diaphragm. During the first week in the hospital the stools gradually became less tarry, and two days before admission to this hospital the patient was allowed out of bed. Twelve hours before admission the patient received an enema, after which he became weak and passed a tarry stool. He received 1000 cc. of blood at that time, and while being transferred to this hospital on the following morning was given another 1000 cc. of blood.

The past history revealed moderate to heavy intake of alcohol until fifteen years before admission, and one drink per week since then. Teeth had been extracted six months and one month prior to admission. The blood pressure was known to be 140 systolic, 80 diastolic on a previous physical examination.

Physical examination revealed a pale man with a warm skin, who was not in shock. The heart and lungs were normal. The abdomen was mildly distended and tympanitic, but no masses could be felt. Peristalsis was quite active.

The temperature was 101°F., the pulse 140 and the respirations 24. The blood pressure was 114 systolic, 60 diastolic.

The nonprotein nitrogen was 55 mg. per 100 cc., the urine normal and the hemoglobin 7 gm. per 100 cc.

The patient was given 3800 cc. of blood during the first night without elevation of the hemoglobin. On gastric suction the return was at first clear, but brown guaiac-positive material was obtained later. During the night he had six or seven large tarry stools mixed with dark blood. On the following day he passed about 400 cc. of whole blood. The hemoglobin at this time was 5.5 gm. Two thousand cc. of blood was given, and an operation was performed.

Differential Diagnosis

Dr. William C. Quinby, Jr.*: This was a straightforward history of a patient with massive intestinal bleeding, as the first sentence said. The question is, of course: What was the source of the bleeding? It is interesting that most of the papers on massive bleeding deal with its management and not with detection of the source. Moreover, there are very few physicians who wish to talk about management in the absence of a positive diagnosis. Statistics are resorted to quite frequently in determining the source. The predominant source of massive gastrointestinal bleeding appears to be a duodenal ulcer. I think that is the presumptive diagnosis in this patient, that it was the presumptive diagnosis when he was admitted to the other hospital and that I must find some evidence in order to depart from it. The evidence in the history and the previous findings mainly supported a diagnosis of duodenal ulcer.

To begin with, this man had had no previous illness. Two months before admission he had some gnawing upper-abdominal distress that was not pathognomonic but was compatible with upper-gastrointestinal ulceration. One point, which was really a negative finding, was that he had never bled before. I should say that that was somewhat against lesions of the hemangiomatous or vascular type. Furthermore, the paucity of symptoms is decidedly against a foreign body or some type of long-standing lesion that might have been present without his knowing it.

There were very few positive physical findings, with the exception of the slight tenderness in the lower part of the midepigastrium noted in the other hospital. By the time the patient arrived at this hospital the gastrointestinal tract was full of blood, and he had distention, which made a diagnosis from the palpation of masses difficult. However, on the previous examination he had no masses. The history gave me the greatest lead for the type of bleeding. This man's bleeding was apparently slow and insidious in onset. He first became dizzy and then passed so much blood that he collapsed at work. He responded briefly to the ulcer regimen in the other hospital, and then he bled so massively that I believe operation was undertaken as a lifesaving procedure. This again is entirely compatible with and characteristic of the silent type of duodenal-ulcer hemorrhage.

The people who advocate conservative management in these cases usually have a diagnosis to

*Assistant in surgery, Massachusetts General Hospital.

proceed on. The methods of obtaining this diagnosis are largely based on x-ray findings. The clinician, however, can do some other things. By passing a sigmoidoscope and a gastric tube or gastroscope he can try to locate a source of the bleeding. The sigmoidoscope and gastroscope were not passed here, but the Levine tube was passed on two occasions, and I think that it was significant that in the other hospital there was no blood in the stomach despite the patient's losing the equivalent of a liter a day. The stools were tarry and were not described as being frankly bloody until later in the course. I doubt whether sigmoidoscopy would have added much to the picture. Anyone bleeding from the lower bowel would pass large quantities of easily recognizable bright blood. Therefore, from the history, the physical findings, the Levine-tube examination and the character of the bleeding, I think I must place the lesion distal to the pylorus and proximal to the ileocecal valve. As I said at the beginning, a silently bleeding duodenal ulcer is by far the most common cause.

I think I should ask Dr. McCort to show me the x-ray films for confirmation of the presumptive diagnosis.

Dr. James J. McCort: The x-ray films do not confirm the presumptive diagnosis. They were sent to us from the other hospital, and we do not have the advantage of a fluoroscopic observation in basing our decision upon them. Barium-enema examination discloses no evidence of a lesion in the colon. The examination of the upper gastrointestinal tract reveals a normal esophagus with some curling in the lower third but no evidence of varices. The stomach and duodenum appear to be normal. Unfortunately, no spot films were made with pressure on the duodenal cap. A film of the chest made at the time the patient was admitted to this hospital shows no abnormality except for a slight prominence of the pulmonary artery. There is no evidence of free air beneath the diaphragm.

Dr. Quinby: You did not do this examination, but had these films submitted to you; how often would you say a duodenal ulcer could be present with shadows like those in the films submitted?

Dr. McCort: I do not know how often, but the possibility must be considered. In our department an examination for ulcer is considered incomplete unless the patient can be moved to the necessary positions and the stomach and duodenum palpated and compressed. All I can say is that I can see no ulcer here.

Dr. Quinby: These were done on the fourth day in the other hospital. Do you agree with the opinions in the literature that if one waits a week or ten days he may miss a bleeding ulcer — that the gastrointestinal series may be normal?

Dr. McCort: Yes, I have had the same experience. The ulcer may not be demonstrable after an interval of treatment.

Dr. Quinby: Dr. McCort has confirmed the statements in the protocol that these x-ray films are essentially noncontributory.

In the Radiology Department of this hospital it has been emphasized time and again that in order to discover a bleeding duodenal ulcer one must make the examination in the acute phase of the ulceration. The maneuver here has been to wait until the patient's condition has become stabilized and then go ahead with the x-ray examination. I think that, country-wide, 25 per cent of the so-called bleeding ulcers have been missed by delayed gastrointestinal series. In this case the negative gastrointestinal series would not exclude a bleeding duodenal ulcer. Indeed, everything points to that! And just as that is the most obvious diagnosis, it is also certainly wrong.

The other causes of bleeding between the pylorus and the ileocecal valve are many. The question that confronted the surgeon was not What caused the bleeding, but Where was the bleeding? I do not believe he had any more information than I have, and I believe the operation was undertaken as a lifesaving measure because the chance of being able to stop this bleeding was excellent, no matter what the cause. Other causes of intestinal hemorrhage without concomitant symptoms of intestinal obstruction are intraluminal or extraluminal neoplasms, some inflammatory granulomas, foreign bodies, diverticula — things of that sort. Polyps and leiomyomas are commonest. In a very rare case, infiltrative tumors in the bowel such as Hodgkin's disease or lymphoma may bleed. Their diagnosis requires a small intestinal series.

I think the best I can say is that this patient probably had a duodenal ulcer on the posterior wall that had bled massively and had eroded the superior pancreatoduodenal artery. In the absence

of that obvious lesion, I would think a benign tumor of some kind in the small bowel was the proper diagnosis.

DR. TRACY B. MALLORY: Dr. Welch, would you tell us your preoperative opinion and findings at operation?

DR. CLAUDE E. WELCH: This patient was first seen by Dr. Perry Culver in an out-of-state hospital. Dr. Culver cannot be here today, to tell you about it. He was the physician who had the nasal tube inserted and the x-ray films taken. At that time he presumed that he was dealing with a bleeding duodenal ulcer. He was somewhat shaken by the fact that no blood was found in the return from the Levine tube, and that fact became more important when the x-ray films taken a few days afterwards were negative, — particularly when the pylorus appeared to be patulous. When the patient did poorly after that and apparently was going to die, Dr. Culver had him brought here, where a nasal tube was again passed, revealing only this bit of guaiac-positive material. As you know, Meulengracht* pointed out a few years ago that patients who die of bleeding duodenal ulcer nearly always have hematemesis in the final stage; therefore we were somewhat inclined to believe that this man did not have a duodenal ulcer and that a small-bowel tumor was more likely. Incidentally, in this hospital, despite Dr. McCort's modesty, the examinations that have been carried on in these difficult conditions have given positive diagnoses in about 85 per cent of the cases, so the x-ray study has been quite an accurate method of diagnosis. We did not have time to do any of those tests on this patient. We had to operate soon after he came in.

CLINICAL DIAGNOSIS

Tumor of small bowel.

DR. QUINBY'S DIAGNOSIS

Bleeding duodenal ulcer.

?Benign tumor of small bowel.

ANATOMICAL DIAGNOSIS

Leiomyoma of small bowel, with massive hemorrhage.

*Meulengracht, E. Fifteen years' experience with free feeding of patients with bleeding peptic ulcer: fatal cases. *Arch. Int. Med.* 80:697-708, 1947.

PATHOLOGICAL DISCUSSION

DR. WELCH: It took about two minutes to make the diagnosis and about twenty minutes to cure the patient. The incision was made considerably lower than would have been safe if we had believed he had a duodenal ulcer. At about the junction of the ileum and jejunum was found a walnut-sized tumor hanging from the edge of the serosa of the bowel and apparently penetrating into the mucosa

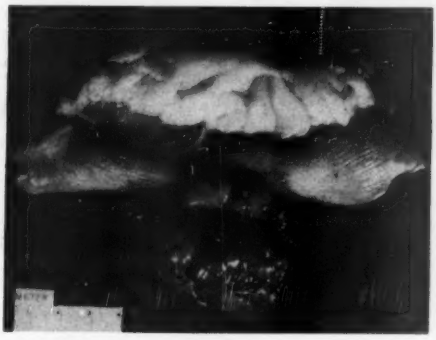

FIGURE 1.

(Fig. 1). It was of some interest to find the tumor at this level, because this man had had definitely tarry stools. On finding this tumor attached to the lower jejunum, I did a resection of the area. There were no enlarged regional lymph nodes, and we thought it was a benign tumor.

DR. QUINBY: Did you find an ulcer, too?

DR. WELCH: Everything else was negative. We explored the stomach and duodenum quite carefully and found no evidence of ulcer.

Dr. Quinby asked me what my preoperative diagnosis was. It was one of those times when everybody was so busy that it was not recorded. The anesthetist asked me my preoperative diagnosis at about the time the abdomen was ready to be closed. Dr. Culver, however, committed himself on the record ahead of time; he believed that a small-bowel tumor was the most likely diagnosis.

DR. MALLORY: The photograph (Fig. 1) shows the resected segment of small bowel, with the almost spherical lobulated tumor presenting exter-

nally from the antimesenteric surface of the gut. The next picture (Fig. 2) shows the internal surface of the intestine, with a small red spot that on closer examination was seen as a relatively fresh thrombus overlying an eroded artery. The microscopical section (Fig. 3) through the tumor and the mucous membrane showed, just at the base of the mucosa overlying the tumor, a small artery

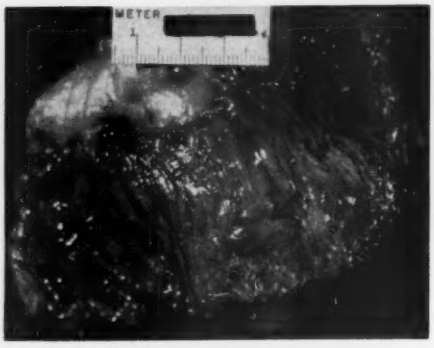

FIGURE 2.

with a localized verruca of its wall, with fresh thrombus overlying the area of erosion at one point. The tumor was obviously a spindle-cell type, the kind of tumor about which opinions frequently differ concerning whether it should be classified as a myoma or a neurofibroma. It was my belief that it was a myoma. It was slow-growing, and there was no evidence of malignancy.

DR. QUINBY: You say the absence of blood in the aspirating tube is a strong indication of the absence

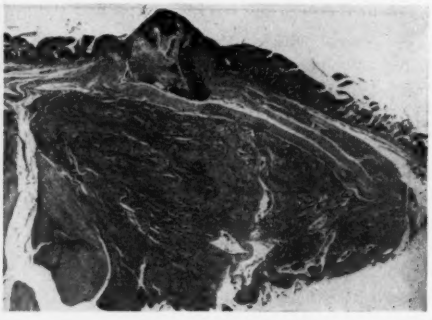

FIGURE 3.

of duodenal ulcer. Of course, at the end stages this man was bleeding massively, and he had a little blood in the tube — not much. There is a great deal of emphasis on that, but is that alone strong evidence?

DR. WELCH: I believe that it is.

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MEDICAL APTITUDE

Twice a year, in May and November, some thousands of college students throughout the country take the Medical College Admission (aptitude) Test. A rating of less than 500 out of a possible 800 considerably lessens their chances (at best about 1 in 3) of being admitted to medical school.

Few if any schools depend on the results of this test to the exclusion of such other important qualifications as good grades, character recommendations and perhaps personableness. Still, the test counts. It counts especially if one doesn't do well at it. And the average dean probably considers it desirable to find a place for the student who rates a straight 600 or 700.

The test is given by the Educational Testing Service of Princeton, New Jersey, the same organization that recently was about to take over the job of helping Selective Service pick the brains to be deferred from military training. The test is divided into four groups: tests of verbal ability (comprehension of English), quantitative ability (simple mathematics), understanding of modern society and basic science. An English professor, a mathematician, a sociologist or a physical chemist would find the questions in his own field puerile. It is doubtful, however, if an average representative of any of these groups, if over 50 years of age, could be sure of getting even a 500 rating in any field but his own, and it is definitely doubtful that a practicing physician of the same vintage could do well in more than one of the groups, if in any of them.

Here are some sample questions for the curious minded:

A sodium atom and a sodium ion (a) contain the same number of electrons; (b) have identical nuclei; (c) are isotopes; (d) have the same physical properties; or (e) differ in the number of protons they contain. Mark the right answer with the electric pencil, Doctor, and let's see how apt you are at your profession. And how many of you are still able to divide 4.80×10^{-10} by 5.27×10^{17} and get the right answer? Or perhaps you would like to decide whether splendid, loquacious, cautious, auspicious or adventurous is the best synonym for *audacious*.

If the surgeon to whom you delegate the job of taking out your child's tonsils and the local practitioner who administers your quarterly shot of penicillin are a bit foggy about these matters, why pester the youngsters with a lot of similar teasers?

The answer is, probably, that the lad who does well in the sort of courses that enable him to take such questions in his stride will be more likely to develop into a good physician than the one who elects courses in Romance philology or political science. Possibly it is not the boy or girl who can write a brilliantly spontaneous essay on sociological trends under the Tudors, but the one who is able only to report laboriously the beheadings and chicaneries of the era with names and dates in order, who will be less likely in later years to con-

fuse acute appendicitis with lumbago or a merthiolate reaction with a fungus infection of the skin, or less likely to require twenty years to perceive the practical significance of such phenomena as the inhibitory effect of certain molds on plate colonies of streptococci.

The classes entering medical school in the fall of 1951 will be filled with young men and women who know considerably more about the physical structure of the universe than their fathers. If their perspicacity, judgment and honesty are no worse, there may be a good future in store.

"VIRGINIBUS PUERISQUE"

IT WILL remain difficult — and at this moment is certainly impossible — to measure the tangible accomplishments of the Mid-Century White House Conference on Children and Youth. However, the recently published testimony of Drs. Paul W. Beaven, Edward B. Shaw and Warren R. Sisson as representative of the opinion of the American Academy of Pediatrics leaves little doubt that the collective effort of the 100,000 or more persons who participated in the preparation for the conference produced significant results.

If physicians — and particularly pediatricians — had expected to be the guiding spirits of the movement, they were due for a surprise. Indeed, one of the incidental unmeasurable accomplishments of the conference was the revelation to the physicians of America of the extent to which the welfare of children is an active concern of educators, clergy, laborers, legislators, lawyers, social workers and responsible citizens of every estate, creed and race. This was not a conference that belonged to or was even led by any group or special interest. Least of all was it a physicians' conference. Not that the doctor was omitted — he was merely "hardly recognizable," as Dr. Sisson put it, "in all this melee. . . . The medical men in attendance learned or relearned one important fact; namely, that important as they may be in the field of improvement of child health, they are neither the sun nor the moon in the heavens, but just one of the stars." This apparently had the sobering effect on the medical observers of helping them to see things in true perspective without making them feel slighted. Dr. Beaven, president of the Academy, came away with a tersely worded impression: "What did I get from this Conference? Many people are dead in earnest that the child shall have his rights. The pediatrician is important, but he has not the weight I thought he had."

Political issues like compulsory health insurance were not introduced on the floor of this conference, — devoted as it was to the problems of healthy children, not sick ones. Most of its 66 recommendations are concerned with the training of personnel who deal with the developmental phases of childhood, parenthood, education, economics, social work, recreation, religion and the problems associated with minority groups. Health is today recognized as more than the absence of disease, and the field of health is not the exclusive concern of any one profession or specialty, whether that be medicine, social work, education, government or diplomacy. Health is an individual and — more importantly in the last half of the twentieth century — a social entity. Custom, usage and necessity have resulted in an undue worldwide preoccupation with disease. Personal health, national health and world health in terms of a balanced global ecology are the great challenge of the second millennium of the Christian Era.

Today's children and youth are the resources of the future. To paraphrase Dr. Edward B. Shaw's summary, the White House Conference dealt with their mental, emotional and spiritual health, not directly but as "a ferment, the results of which may finally be apparent even though they cannot now be precisely defined. It is impossible for me to witness this effort in any spirit of cynicism or to fail to appreciate the depth of feeling for children which permeated this entire effort."

A COMMON GOAL

STATE medical-society membership apparently has little appeal for graduates in medicine who are not engaged in the clinical practice of their profession; at least, this seems to be the case in Massachusetts, despite the goal that all physicians have in common. The most recent information available

concerning one of the three medical schools in the Commonwealth enables the following figures on the Society membership of physician teachers in certain of the science departments to be presented.

In the Department of Anatomy only 2 of the eligible teachers in the 1950 roster were members of the Society,—at least in the first months of the year, — and 6 had not joined; 3 of the physiologists had joined as compared with 13 who were not members; in chemistry 2 had joined and 19 had not; in bacteriology the comparable figures were 6 and 12. Only in pathology did the joiners outnumber the nonjoiners, 24 to 23 — perhaps because so many pathologists, being somewhat closer to the patient, are members of hospital staffs.

A similar although not entirely comparable situation apparently exists in regard to the teaching of public health, if not to its practice. A prominent school of public health was subjected to the same general analysis, the teaching personnel being arranged according to rank rather than by the specialty taught. In this study it was observed that of 5 eligible emeritus professors 3 were society members and 2 were not; 3 of the professors were members and 4 were not; neither of the 2 associate professors had joined, having paired off, apparently with the 2 clinical professors, both of whom were members; the 2 lecturers were also paired, 1 in favor of society membership and 1 against it; 7 of the assistant professors were members and 4 were not; the only associate was a member and 12 of the instructors had joined, as against 4 who had not. Two of the 3 assistants were members, and none of the 11 research associates and fellows had joined. These transient residents, however, should hardly be counted.

The scores stand with 37 medical-science teachers having joined the Society and 73 having held back, and 31 public-health teachers and research personnel who have joined and 28 who have stayed out. Perhaps fortunately, in the school of public health, it is the younger men of lower rank who have most appreciated the desirability of solidarity within the profession.

The presentation of these simple statistical data implies no criticism of the physicians in the categories mentioned who have not joined a Society whose requirements for membership were originally designed for the practicing clinician. The most onerous of these requirements, so far as the full-time investigator or teacher is concerned, is that a candidate must be licensed to practice medicine within the United States or its territories; another is that he must be examined in clinical subjects. Nonmembership in the Society, moreover, does not restrict the opportunities for work or advancement of the teacher outside the hospital or the worker in the laboratory as it does those of the practitioner; this powerful incentive to join is lacking.

All types of physicians are losers, however, if certain groups find it more difficult and less to their advantage to join their medical societies than do others. Fences should be torn down and not erected; the exchange of ideas should be facilitated and not impeded. Perhaps, in view of the diversity of the activities of modern medicine, the rules should be changed to give more nearly equal opportunities to the various nonpracticing specialties.

PRIZE ESSAY AWARDS

THE editors of the *Journal* take pleasure in announcing that the third annual prize essay contest for fourth-year medical students has been won by John A. Meyer, a senior student at Western Reserve University School of Medicine. Mr. Meyer's report and discussion of a case of "Coarctation of the Aorta," which was awarded the first prize of $100, will be published in a subsequent issue.

Robert R. Simpson, a member of the fourth-year class at Indiana University School of Medicine, has been awarded the second prize, a two-year subscription to the *Journal*. The title of his report was "A Study of Aneurysm of the Thoracic Aorta."

The competition this year was based on the report of a case that the student had personally studied, with a discussion and review of the subject. This has proved to be the most popular assignment so far, and the number of competitors was nearly double that of the preceding year. The same subject will be assigned for the 1952 contest —

a case personally studied by the student, with a discussion and review of the literature — manuscripts to be in the hands of the editor by March 15, 1952.

It is not necessary to dwell on the satisfaction that the editors derive from the student interest in the *Journal*. Recent mention has been made of the fact that on March 22 the number of student subscribers reached 5000, and has now gone somewhat beyond that figure.

A new edifice is to be erected in Cincinnati, during the present season, for the Medical College of Ohio. Its style is to be the domestic gothic, *and it is intended to be not inferior to the best in the country.*

Boston M. & S. J., *May 21, 1851*

OBITUARY

DOUGLAS ARTHUR THOM

1887–1951

Dr. Douglas Arthur Thom was born in Boston, on October 4, 1887. His early years, however, were spent in Camden, Maine, which he regarded throughout his life as his real home. He was graduated in 1912 from the University of Vermont Medical School, from which he also received the honorary degree of M.S. in 1923. He entered the field of neuropsychiatry as a staff member of the Monson State Hospital, later working as assistant pathologist in the Massachusetts Department of Mental Health (then known as the Department of Mental Diseases) under Dr. Elmer E. Southard, with whom he published, among other studies, "An Anatomical Search for Idiopathic Epilepsy." Of considerable importance were his three papers on the role of heredity in epilepsy, which shed doubt on the current assumption that the disease was inherited. He was a pioneer in introducing the modern treatment for paresis, which he carried out at the Grafton State Hospital.

At the close of World War I, after a long Army service in France, where he had been particularly successful in treating neuropsychiatric cases, he became active in setting up neuropsychiatric clinics for the Red Cross, but he soon entered on his greatest project, the now world-famous Habit Clinic for Child Guidance. He was one of the first to see the relation of behavior disorders and nervous and mental disease to faulty habits in children of preschool age. His advice was widely sought, and his papers and books were translated into many languages. Although he had a rare talent for dealing with individuals, he also had organizing ability of the first order. He soon developed a large private practice with problem children (and "problem parents," as he often said), but he fulfilled a national function as a co-ordinator of research and a stimulator of organized effort.

Dr. Thom was for more than twenty-five years a member of the Massachusetts State Department of Mental Health; he was an officer of the New England Society of Psychiatry, the Boston Society of Psychiatry and Neurology, the Massachusetts Psychiatric Society and many others. He was an honorary member of British and French neuropsychiatric societies.

He was for many years professor of psychiatry at Tufts College Medical School, and had also been instructor in psychiatry at Harvard Medical School and professor of psychiatry at the University of Vermont. He was one of the original faculty of the Smith College School of Social Work.

During the last war he was a Colonel and served as a consultant to the Surgeon General and to the Army Headquarters at New York. Although he was stricken with a known fatal disease and retired from the Army thereafter, he immediately entered into one of the most active periods of his life, as consultant to the Army, the Veterans Administration and the Massachusetts Department of Mental Health.

He met his illness with equanimity; the day before his death he visited a veterans' hospital as consultant. With all his greatness in medicine, he was, perhaps, greatest in his capacity for friendship. Far and wide, those who knew him loved him, and each one of us felt a closeness that transcended all official and professional concepts. All ranks and types of people used the name "Doug" with affection and respect.

He died on February 23, 1951. He is survived by his widow and a son.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

JACK — Frederick L. Jack, M.D., of Boston, died on May 3. He was in his ninety-first year.

Dr. Jack received his degree from Harvard Medical School in 1884. He was formerly aural surgeon-in-chief of the Massachusetts Eye and Ear Infirmary. He was a member of the American Otological Society, the American Laryngological, Rhinological and Otological Society and the New England Oto-Laryngological Society and a fellow of the American College of Surgeons.

There are no immediate survivors.

O'SULLIVAN — Patrick A. O'Sullivan, M.D., of Somerville, died on November 26. He was in his fifty-fourth year.

Dr. O'Sullivan received his degree from Tufts College Medical School in 1926. He was a member of the New England Obstetrical and Gynecological Society.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

ALCOHOLISM — A MEDICAL PROBLEM

"Intemperance in eating and in drinking is a symptom of the psyche." So it was written in the fifth century B.C. by Hippocrates or one of his pupils. However, this idea made very little headway until the last decade and a half, when there suddenly developed in some medical and lay circles a trend toward recognizing alcoholism as a disease or, more correctly, a medical syndrome. Experimental findings have begun to replace intuition and clinical activities are demonstrating that the medical approach differs from the propositions of "reform societies."

In spite of this recent progress there has been no official recognition of this medical problem by any large medical body until, less than a year ago, the World Health Organization placed its official stamp on alcoholism as a public-health problem by forming a special committee on alcoholism, which held its first meeting at Geneva in December, 1950. This expert group, composed of psychiatrists from six nations, believed that alcoholism was a problem of great magnitude in many countries but its seriousness had not been generally recognized. Because of many cultural and social differences, much of the discussion dealt with basic concepts. However, the general conclusion reached was that medicine, along with its allied professions, could make a significant contribution to the welfare of society by attacking the problem of alcoholism on a scientific basis.

At its clinical session in Cleveland, Ohio, in December, 1950, the House of Delegates of the American Medical Association accepted the problem of alcoholism as a responsibility of the medical profession, and a resolution was passed referring the matter to the Committee on Chronic Diseases. This committee has recently created a special subcommittee for the purpose of formulating a comprehensive program for medicine's participation in solving the problems of alcoholism.

Attitudes in the past have relegated the alcoholic "to the limbo of a social pariah with an unwarranted stigma attached." A similar situation previously existed in regard to tuberculosis, cancer and other diseases until the causes and effects of these illnesses were studied. Discussions of these conditions, along with findings relating thereto, were brought into the light so that all could see and learn. These diseases have come to be regarded as recognizable entities, responsive to treatment and preventable in many instances by early detection and prompt therapy. The medical profession, possessing specialized knowledge and skills, can and should turn its energies toward developing ways and means of alleviating and preventing this scourge of mankind.

JOHN C. AYRES, M.D., *Director*
Division of Alcoholism

CORRESPONDENCE

SUSPENSION OF LICENSE

To the Editor: At a meeting of the Board of Registration in Medicine on April 26, 1951, it was voted to suspend for one year the registration of Dr. Sidney S. Cahan, 55 Ridgewood Street, Dorchester.

ROBERT C. COCHRANE, M.D., *Secretary*
State House
Boston

REVOCATION OF LICENSE

To the Editor: At a meeting of the Board of Registration in Medicine held April 26, 1951, it was voted to revoke the registration of Dr. James W. Elliott, 359 Massachusetts Avenue, Boston.

ROBERT C. COCHRANE, M.D., *Secretary*
State House
Boston

BOOK REVIEW

Vernal Conjunctivitis. By M. N. Beigelman, M.D. With a foreword by Sir W. Stewart Duke-Elder, K. C. V. O., M.A., D.Sc. (St. And.), Ph.D. (Lond.), M.D., Ch.B., F.R.C.S., Hon. D.Sc. (Northwestern). 8°, cloth. 430 pp., with 49 illustrations. Los Angeles: University of Southern California Press, 1950. $6.00.

With the authority of 712 citations from the world's literature, and documented by penetrating clinical and histologic studies of 236 of his own patients, Beigelman uses 430 pages and 49 illustrations to admit that definite etiology and useful therapy are imperfectly understood in vernal conjunctivitis. In spite of this, he exposes to the clinical and research ophthalmologists the present status of knowledge about vernal conjunctivitis.

Vernal conjunctivitis is a bilateral affliction, with incidence running as high as 1 per cent of all eye diseases, and with essentially negative bacteriologic findings. There are two main types: the bulbar type, which produces a gelatinous pericorneal ring of tissue, and the palpebral type, which shows congestion of the conjunctiva of the lids, with vascularized follicles that crowd each other to give a cobblestone effect (Arlt, 1846; Desmarres, 1855; von Graefe, 1870; and others). Trantas (1910) in a critical review emphasized the fact that there is corneal involvement in half the cases, best found with the corneal microscope and vital staining, as learned since 1911 by means of the slit-lamp. Herbert (1903) demonstrated a universal increase in eosinophils of the blood and in the conjunctival scrapings in vernal conjunctivitis and keratitis.

Subjectively, the outstanding symptom is intense and persistent itching in most of the victims. A sensation of foreign body in the eye, burning sensation and photophobia may stem from the corneal lesions.

The seasonal incidence, the follicles, the itching and the eosinophilia suggest allergy, but no specific allergen has yet been demonstrated in spite of the vast amount of investigation that has been done along these lines.

The crowning paradox comes with therapy of vernal conjunctivitis and keratitis. In spite of intensive therapy with astringents, antihistaminics and Cortone, the affliction blithely runs its course, through periodic and seasonal recurrences, for five to six years. Then, for no known reason, the affliction completely disappears, leaving a clear and normal cornea and conjunctiva.

Since this monograph is the latest word on the subject of vernal conjunctivitis and keratitis, a copy should have a place in the working library of every clinical and research ophthalmologist.

J. HERBERT WAITE, M.D.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Psychiatric Sections in General Hospitals. By Paul Haun, M.D., Med.Sc.D., assistant professor of psychiatry, Georgetown University School of Medicine. 4°, cloth, 80 pp., with 15 plans. New York: F. W. Dodge Corporation, 1950. $4.00.

In this semi-atlas an analysis of eight plans of psychiatric sections in general hospitals is presented, and they are rated from excellent to unsatisfactory, according to their desirable and undesirable features. In conclusion, Drs. Charles Butler and Addison Erdman present a suggested plan for a small psychiatric section of 24 beds in a general hospital of about 200 beds. In the preliminary text Dr. Haun discusses the question in general. The text is concluded with a bibliography confined to the subject, and there is a good index. The small volume should be in all special collections on psychiatry and hospitals.

The Cerebral Circulation in Health and Disease. By Carl F. Schmidt, M.D., Laboratory of Pharmacology, University of Pennsylvania School of Medicine. Edited by Robert F. Pitts, M.D., Ph.D., professor of physiology, Syracuse University College of Medicine. 8°, cloth, 78 pp., with 12 illustrations. Springfield, Illinois: Charles C Thomas, 1950. $2.00. (Publication No. 68, *American Lecture Series.*)

In this small monograph the author discusses briefly the anatomy of the cerebral arterial system, the physiology of the cerebral circulation, its relation to cerebral function and its status in health and disease in man.

The Physiological Basis for Oxygen Therapy. By Julius H. Comroe, Jr., professor of physiology and pharmacology, University of Pennsylvania School of Medicine, and clinical physiologist, Hospital of the University of Pennsylvania; and Robert D. Dripps, professor of anesthesiology, University of Pennsylvania School of Medicine, and director of anesthesiology, Hospital of the University of Pennsylvania. Edited by Robert F. Pitts, M.D., Ph.D., professor of physiology, Cornell University Medical College. 8°, cloth, 85 pp. Springfield, Illinois: Charles C Thomas, 1950. $2.00. (Publication No. 42, *American Lecture Series.*)

The authors in this small monograph present the rational, physiologic basis for oxygen therapy, and conclude that the major field of its usefulness is anoxia. There are chapters on the inhalation of oxygen in normal man, the etiology, diagnosis and treatment of anoxia in its many forms, the use of oxygen in conditions without anoxia, and the possibilities of harm from the inhalation of oxygen.

Morbus Caeruleus: An analysis of 114 cases of congenital heart disease with cyanosis. Edited by E. Mannheimer. 8°, cloth, 332 pp., with 122 illustrations. Basel: S. Karger, 1949.

This Swiss monograph is the joint work of eleven authors. It covers the whole disease from the embryology to treatment. The concluding chapter lists a number of representative cases out of a total of 104 cases. There is an extensive bibliography and a good index. The publishing is excellent, including the reproduction of the radiograms. The monograph should be in all collections on diseases of the heart.

Elements of Medical Mycology. By Jacob H. Swartz, M.D., assistant professor of dermatology, Harvard Medical School. In collaboration with Ethel M. Rockwood, M.D., dermatologist, Massachusetts General Hospital. With an introduction by Fred D. Weidman, M.D., professor of dermatologic research, University of Pennsylvania. Second edition. 8°, cloth, 241 pp., with 138 illustrations. New York: Grune and Stratton, 1949. $5.50.

This second edition of a manual first published in 1943 has been revised and brought up to date. Clinical aspects are discussed in more detail and are more fully illustrated. A glossary of special terms concludes the text. There is a good index and a valuable chart of the pathogenic fungi.

Fortschritte der Tuberkuloseforschung. Vol. III. Edited by H. Birkhauser and H. Bloch. 8°, paper, 307 pp. Basel: S. Karger, 1950. (*Bibliotheca Tuberculosea*, Fasc. 3.)

This volume contains seven progress reports on various aspects of tuberculosis. Three of the authors are from the United States. The summaries are comprehensive in scope and well written. Each report concludes with a long bibliography. The subjects reviewed are the chemistry of tuberculin, streptomycin resistance of the Koch bacillus, the operative treatment of pulmonary tuberculosis, advances in bacteriologic research, hereditary factors in tuberculosis of man and animals, allergy during the course of latent tuberculous infection and chemotherapy. The articles are written in English, French or German. The volume is well published and should be in all collections on the subject.

Pocket Encyclopedia of Atomic Energy. Edited by Frank Gaynor. 8°, cloth, 204 pp., with illustrations and tables. New York: Philosophical Library, 1950. $7.50.

The material in this encyclopedia is arranged in alphabetical form and should be found in all libraries. It comprises a collection of brief explanations and definitions of terms used in the fields of nuclear physics and atomic energy. The comprehensive table of the isotopes is especially valuable.

Textbook of Endocrinology. Edited by Robert H. Williams, M.D., executive officer and professor of medicine, University of Washington Medical School, Seattle. 8°, cloth, 793 pp., with 168 illustrations. Philadelphia: W. B. Saunders Company, 1950. $10.00.

This composite treatise is the joint work of twelve specialists in their particular fields. The disorders of the various glands are discussed at length, including the pancreas and its relation to diabetes mellitus. There are also special chapters on the influence of the endocrine glands on growth and development, neuroendocrine and psychodynamic aspects of the endocrinopathies, obesity, and laboratory diagnostic and assay procedures. Lists of selected references are appended to the different chapters and there is a good index. The publishing is well done. The book is recommended for all medical libraries and interested physicians.

Nutrition in Health and Disease. By Lenna F. Cooper, M.A., M.H.E., Sc.D., consultant dietitian, Montefiore Hospital, New York City; Edith M. Barber, M.D., writer and consultant, *Food and Nutrition*, and food editor, *General Features Syndicate*; and Helen S. Mitchell, Ph.D., dean of the School of Home Economics, University of Massachusetts. Associate author, Henderika J. Rynberger, M.S., assistant professor of science, Cornell University–New York Hospital School of Nursing. Eleventh edition. 8°, cloth, 744 pp., with 133 illustrations. Philadelphia: J. B. Lippincott Company, 1950. $4.00.

This standard textbook, first published in 1928, has been completely revised in this new edition. The text is divided into four parts: principles of nutrition; diet in disease; food selection and preparation; and tables and special tests. There is a comprehensive index, and the volume is well published. It should prove valuable as a reference work for all libraries, general and medical, and to all physicians and other persons interested in nutrition.

Clinical Electrocardiography. By Francis F. Rosenbaum, M.D., assistant clinical professor of medicine, Marquette University School of Medicine, member of staff, Milwaukee County Hospital, member of associate staff, Columbia Hospital, member of adjunct staff, Milwaukee Children's Hospital, and cardiac consultant and attendant, Cardiac Clinic, Milwaukee Children's Hospital, Milwaukee. Edited by Henry A. Christian, A.M., M.D., LL.D., Sc.D. (Hon.), M.A.C.P., Hon. F.R.C.P. (Can.), and D.S.M. (A.M.A.). 8°, cloth, 201 pp., with 82 illustrations. New York: Oxford University Press, 1950. $4.50. (Reprinted from *Oxford Loose-Leaf Medicine* with the same page numbers as in that work.)

This monograph on clinical electrocardiography is now made available to physicians separate from the large *Oxford Loose-Leaf Medicine.* The subject is brought up to date, and there is a bibliography of 211 titles. A good index has been provided.

(*Notices on page xiii*)

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Gray, L.: J. Clin. Endocrinol. 3:92 (Feb.) 1943.

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Head Pain as a Diagnostic Lead

Frequently the presence of head pain is overlooked. The physician learns of it only if he has made an effort to elicit the information. Since the etiology of the pain is the basis of rational management, the patient should be warned against taking medication before diagnosis is made.

Friedman[1] deplores the tendency to call any chronic recurring headache migraine. Careful history-taking and full physical and neurological examinations are essential for accurate diagnosis. A good starting point is a description of the headache — its character, laterality, frequency and intensity.[2]

The following chart gives briefly the primary diagnostic leads and treatment for the most common types of headache.

Etiology of Headache	Primary Diagnostic Data	Primary Therapy
Inflammatory e.g., Meningitis Abscess	Inflammation of intracranial structures; fever; leucocytosis; bacteriologic diag.	Specific: sulfonamides and antibiotics. Symptomatic: analgesics.
Tumor	Pain varies as spinal press. changes; skull X-ray.	Specific: surgery. Symptomatic, analgesics &/or hypnotics.
Sinusitis	Sinus congestion and infection; cloudy X-ray.	Specific: antibiotics and drainage. Symptomatic: analgesics.
Hypertensive	Hypertension present but pain not related to b. p. level; Dihydroergotamine. relieves pain.	General hypertension therapy; sedation. Symptomatic: analgesics.
Migraine & other vascular headaches	Headache: recurrent, intense, throbbing. No organic causation; migraine in family; patient: energetic, perfectionist. Visual prodromata; g-i. upset during headache.	To abort attack: oral ergotamine plus caffeine. General: adjustment to minimize nervous stress.

Data here tabulated is from: Wolf, G., Jr.,[3] and Friedman, A. P.[4]

Cecil[5] ranks vascular headaches, e.g., migraine and tension headaches, as the most commonly encountered of all. Because of their functional nature and usual recurrence at frequent intervals, they present a long-term therapeutic problem.

Therapy is conducted along two lines:

1) Psychotherapy to reduce the frequency of attacks. This consists mainly of advice on emotional adjustment to stressful situations and guidance toward a good balance between work and relaxation.

2) Treatment of the distressing attack to prevent the usual period of incapacitation. Many investigators have reported that ergotamine preparations are effective for relief of the acute migraine attack in 80% of cases.[1,6] *The drug is given immediately when an attack is approaching and dosage adjusted to the needs of the individual.*

1. Friedman, A. P. and von Storch, T.: 99th A.M.A. Session, June 1950. 2. Butler, S. and Hall, F.: M. Clin. N. Amer., p. 1439 (Sept.) 1949. 3. Wolf, G., Jr.: M. J. 54:25, 1951. 4. Friedman, A. P. and Conn, H. T.: Current Therapy, 1950, p. 563; Saunders Co., Phila. 5. Cecil, R. L.: A Textbook of Medicine, ed. 7, 1948, p. 1483; Saunders Co., Phila. 6. Horton, B. et al: Staff Meet. of Mayo Clinic 20:241, 1945.

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NOTICES

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

CONFERENCE PROGRAM
9–10 a.m.

Tuesday, May 22 — Cholesterol and Arteriosclerosis. Dr. John A. McGowan, Jr.

Wednesday, May 23 — Physio-pathology of Platelets. Dr. Mario Stefanini.

Friday, May 25 — Cardiovascular Abnormalities in Some of the Common Infectious Diseases. Dr. Louis Weinstein.

Tuesday, May 29 — X-ray conference. Dr. Alice Ettinger and Staff.

Wednesday, May 30 — Holiday (no conference).

Conferences are held in the Pratt Lecture Hall. All exercises are open to the medical profession.

SOCIETY MEETINGS AND CONFERENCES

MAY 21. New England Cardiovascular Society. Page 741, issue of May 10.

MAY 21–24. Massachusetts Physicians Art Association. Page 742, issue of May 10.

MAY 21–25. International Society of Orthopaedic and Traumatic Surgery. Page 422, issue of March 15.

MAY 22–24. Massachusetts Medical Society. Annual meeting. Hotel Statler, Boston.

MAY 22–29. Joseph H. Pratt Diagnostic Hospital. Notice above.

MAY 23. School Physicians' Association. Page 614, issue of April 19.

MAY 23. U. S. Naval Reserve Volunteer Medical Unit No. 1-1. Page 698, issue of May 3.

MAY 24–26. American Goiter Association. Page 384, issue of March 8.

MAY 28. New England Diabetes Association. Page 742, issue of May 10.

MAY 29. New England Rheumatism Society. Page 742, issue of May 10.

JUNE 6–9. American Proctologic Society. Page 698, issue of May 3.

JUNE 7 AND 8. International Academy of Proctology. Page 742, issue of May 10.

JUNE 7–9. American Radium Society. Page 656, issue of April 26.

JUNE 7–10. American College of Chest Physicians. Page 494, issue of March 29.

JUNE 9 AND 10. American Society for the Study of Sterility. Page 274, issue of February 15.

JUNE 11–15. American Medical Association. Annual Session. Atlantic City, New Jersey.

JUNE 11–15. Physicians' Art Exhibit at A.M.A. Meeting. Page 614, issue of April 19.

JUNE 15–17. American Electroencephalographic Society. Page 346, issue of March 1.

JUNE 18–20. American Neurological Association. Page 614, issue of April 19.

JUNE 20–23. American Orthopaedic Association. Page 422, issue of March 15.

SEPTEMBER 4–8. American Congress of Physical Medicine. Page 238, issue of February 8.

SEPTEMBER 19. American Medical Writers' Association. Page 1066, issue of December 28.

SEPTEMBER 19–21. Mississippi Valley Medical Society. Page 1066, issue of December 28.

SEPTEMBER 30–OCTOBER 2. New Hampshire Medical Society. Annual Meeting. Equinox House, Manchester, Vermont.

OCTOBER 31. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.

NOVEMBER 10–21. Pan-Pacific Surgical Association. Page 680, issue of October 26.

JULY 8–11, 1952. Thirteenth British Congress of Obstetrics and Gynaecology. Page xv, issue of December 14.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, MAY 24

THURSDAY, MAY 24

*9:00–10:00 a.m. Grand rounds. Robert Breck Brigham Hospital.
*9:00–10:00 a.m. Grand rounds. House of the Good Samaritan.
*9:00–11:00 a.m. Medical grand rounds. Joseph H. Pratt Diagnostic Hospital.

FRIDAY, MAY 25

*9:00–10:00 a.m. Medical conference. Joseph H. Pratt Diagnostic Hospital.
*9:00 a.m.–12:00 m. Combined medical and surgical staff rounds. Peter Bent Brigham Hospital.
*10:00 a.m. Surgical clinic. South End Health Unit, 57 East Concord Street, Boston.
*12:00 m.–1:00 p.m. Research conference. Dowling Amphitheatre, Boston City Hospital.
*12:00 m.–1:30 p.m. Surgical pathologic conference. Joseph H. Pratt Diagnostic Hospital.
*1:30 p.m. Tumor clinic. Outpatient Department, Mount Auburn Hospital, Cambridge.
*2:00–4:00 p.m. Therapeutic conference. Joseph H. Pratt Diagnostic Hospital.
*4:00–6:00 p.m. X-ray conference. Joseph H. Pratt Diagnostic Hospital.

SATURDAY, MAY 26

*9:00–10:00 a.m. Surgical grand rounds. Joseph H. Pratt Diagnostic Hospital.

MONDAY, MAY 28

*11:30 a.m.–12:15 p.m. Clinical conference. South End Health Unit, 57 East Concord Street, Boston. Dr. Cleaveland Floyd in charge.
*12:15–1:15 p.m. Clinicopathological conference. Main Amphitheatre, Peter Bent Brigham Hospital.
5:15 p.m. Staff meeting. Harris Hall, New England Deaconess Hospital.

TUESDAY, MAY 29

*9:00–10:00 a.m. Medical conference. Joseph H. Pratt Diagnostic Hospital.
*12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.
*1:30–2:30 p.m. Pediatric rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

*Open to the medical profession.

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